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**Facial electromyography (EMG) activity was recorded from the zygomaticus major and corrugator supercilii muscle regions to examine emotion-specific reactivity in 24 currently depressed individuals meeting DSM-IV criteria for Major Depression, Recurrent with Seasonal Pattern, and no other current Axis I diagnosis, and 24 controls with normal mood and no history of depression. Based on models of seasonal affective disorder (SAD) and a proposed role for learned associations between depressive behavior and environmental stimuli signaling low light and winter season, participants were exposed to light- and season-relevant environmental stimuli and were asked to imagine what they would be feeling and thinking if they were actually in the picture. Skin conductance response was also assessed to determine participants' general sympathetic arousal to the stimuli. Results indicated that SAD participants: 1) responded to bright light stimuli with decreased corrugator mean EMG activity relative to low light stimuli; 2) demonstrated no increases in zygomatic mean EMG activity to bright light stimuli; 3) reported an exacerbation of baseline depressed mood following low light and winter stimuli and an improvement in depressed mood following bright light stimuli; and 4) evidenced increased SCR magnitude to bright light stimuli as compared to low light stimuli. Notably, corrugator and self-report mood ratings support previous findings of heightened psychophysiological reactivity and exacerbated depressed mood after exposure to light-relevant stimuli in SAD and suggest that light intensity may be more salient than seasonal cues in determining affective reactivity. Further research is needed to understand how these associations develop, and to establish the clinical implications for psychophysiological measures in SAD assessment and treatment monitoring.**

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## ABSTRACT


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# SURFACE FACIAL ELECTROMYOGRAPHY REACTIONS TO LIGHT-RELEVANT AND SEASON-RELEVANT STIMULI IN SEASONAL AFFECTIVE DISORDER

by

Kathryn Tierney Lindsey

Doctoral Dissertation submitted to the faculty of the

Department of Medical and Clinical Psychology

Graduate Program of the Uniformed Services University

of the Health Sciences in partial fulfillment

of the requirements for the degree of

Doctor of Philosophy, 2005

## DEDICATION

This work is dedicated to the memory of Thomas E. Tierney and Cecilia M. Tierney whose love, kindness, and generosity have inspired every accomplishment in my life.

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# INTRODUCTION

Several studies examining facial electromyography (EMG) as it relates to nonseasonal depression have been conducted over the last 20 years. Surface facial EMG studies have linked facial muscle group activity to specific emotions (Schwartz, Fair, Salt, Mandel, & Klerman, 1976a). Patterns of corrugator muscle activity, associated with a frown, can reliably detect a negative affective state during unpleasant or unhappy imagery in nonclinical and depressed samples. Despite the extensive examination of psychophysiological responding in the nonseasonal depression literature (e.g., Cacioppo, Bush, & Tassinary, 1992; Greden, Genero, Price, Feinberg, & Levine, 1986; Oliveau & Willmuth, 1979; Schwartz et al., 1976a), only two studies have focused on psychophysiological responsiveness in seasonal affective disorder (SAD). Rohan, Sigmon, Dorhofer, and Boulard (2004) provided the first empirical data linking heightened psychophysiological arousal (i.e., skin conductance response magnitude) to seasonality. These researchers compared control participants to individuals with subsyndromal SAD (S-SAD; reverse vegetative symptoms during the winter that are less severe than in SAD). Similarly, Sigmon, Whitcomb-Smith, Boulard, and Kendrew (2002) found increased skin conductance reactivity in seasonal samples. Specifically, participants with SAD evidenced more significant skin conductance responses (SCRs) and greater SCR magnitude when compared to nonseasonal, nondepressed controls. Although increased skin conductance reactivity suggests heightened sympathetic arousal in SAD, other psychophysiological measures, especially those that can distinguish specific emotional responses (i.e., surface facial EMG activity), may have even greater utility in psychophysiological research on SAD.

This paper will accomplish the following: 1) provide a review of basic information concerning SAD (i.e., symptoms, diagnosis, and prevalence); 2) review SAD epidemiology; 3) highlight the empirical status of psychological factors in SAD; 4) describe preliminary psychophysiologic studies conducted with SAD and seasonality samples; 5) review the EMG literature as it pertains to nonseasonal depression; 6) discuss the potential role for surface facial EMG in SAD; and 7) propose a study using facial EMG as an indicator of mood-specific reactivity to light- and season-relevant stimuli in an effort to expand integrative psychological and physiological models of SAD.

## Defining SAD: Symptoms, Diagnosis, and Prevalence

Major Depressive Disorder (MDD) includes the presence of a Major Depressive Episode as outlined in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994). A Major Depressive Episode requires five or more of the following symptoms for at least 2 weeks, with one of the five being either criterion 1 or 2: 1) depressed mood, almost all day, nearly every day; 2) decreased interest or pleasure in activities, most of the day, nearly every day; 3) feeling worthless or guilty, more often than not; 4) loss of energy or excessive fatigue, most of the time; 5) decreased concentration or indecision, more days than not; 6) significant weight loss or gain; 7) most days, observable evidence of significant psychomotor agitation or retardation; and 8) recurrent consideration of death or suicide. Although most symptoms experienced are similar to those of nonseasonal MDD, SAD patients often exhibit a cluster of the reverse vegetative symptoms (e.g., fatigue, excessive sleeping patterns, and overeating; Rosenthal et al., 1984b).

According to DSM-IV (APA, 1994), a Seasonal Pattern Specifier can be applied to Major Depressive Disorder, Recurrent, or Bipolar I or II Disorder when the following occur: 1) a regular temporal relationship has existed between the onset of the MDD and a specific time of the year (e.g., recurrence in the fall or winter); 2) a full remission of the depressive symptoms is experienced at another specific time of the year (e.g., symptoms remit in the spring or summer); 3) for the last 2 consecutive years, two Major Depressive Episodes have occurred that exhibit the temporal relationship defined in criteria 1 and 2, and no nonseasonal depressive episodes have occurred during that time frame; and 4) the seasonal depressive episodes have outnumbered the nonseasonal Major Depressive Episodes over the individual's lifetime. Rosenthal et al.'s (1984b) research criteria for SAD include: 1) a history including at least two Major Depressive Episodes; 2) a minimum of 2 successive years of depressive symptoms that wax and wane with the seasons; 3) absence of any other Axis I disorders; and 4) seasonal variations in mood and behavior that cannot be accounted for by seasonally-changing psychosocial factors (e.g., final examinations for a college sample). The focus of this study is on winter-type SAD, involving Major Depressive Episodes that occur in fall and/or winter and remit in the spring.

***Seasonality Continuum Hypothesis***

SAD symptom expression appears to exist on a continuum of mild to moderate symptomatology. Seasonally-related changes in vegetative functioning (e.g., sleeping too much, decreased energy, and excessive eating including increased intake of complex carbohydrates and sugars) have been noted in the general, nonclinical population (Kasper, Wehr, Bartko, Gaist, & Rosenthal, 1989; Terman, Terman, Quitkin, McGrath,

Stewart, & Rafferty, 1989). Although they did not meet criteria for diagnosis of clinical SAD, some individuals in these community samples experienced mild vegetative symptoms (e.g., increased appetite, sleep duration, and fatigue) during winter, supporting the notion that SAD symptoms lie on a seasonality continuum. Mild to moderate vegetative symptom expression, referred to as S-SAD, is less intense than that experienced in full diagnosable SAD.

According to the seasonality continuum hypothesis (Kasper et al., 1989; Terman et al., 1989), the most severe symptoms are experienced by those with clinical SAD, whereas, at the other end of the spectrum are nonseasonal individuals, those having little to no behavioral variation across the seasons. Along the middle of the continuum lies S-SAD. The number and severity of reverse vegetative symptoms present, including the degree of hyperphagia, hypersomnia, and anergia experienced, are subsumed under the construct "seasonality."

***SAD Epidemiology***

Because SAD research has only recently gained momentum within the psychopathology literature, there are, to date, no large-scale epidemiological studies on SAD available for review. In general, MDD has been found to be highly prevalent in the U.S. population including a lifetime risk of 7 to 12% for men and 20 to 25% for women (U.S. Department of Health and Human Services [USDHHS], 1993a). These rates are comparable to recent results from the National Comorbidity Survey Replication study (NCS-R; Kessler et al., 2003). Based on the Composite International Diagnostic Interview (CIDI; Robins et al., 1988), lifetime risk for MDD was 16.2% (95% confidence interval [CI] = 15.1-17.3), and estimated 12-month risk was 6.6% (95% CI = 5.9-7.3).

Results further indicated a mean depressive episode duration of 16 weeks (95% CI = 15.1-17.3). In addition, using a measure that assesses the worst month of symptoms experienced within the past year, the Quick Inventory of Depressive Symptomatology Self-Report (QIDS-SR), 38.6% of participants reported clinically significant symptoms classified as moderate, 38.0% reported severe symptoms and 12.9% reported very severe symptoms (Kessler et al., 2003).

Another recent study reported a significant cost of lost productivity among U.S. workers with depression (Stewart, Ricci, Chee, Hahan, & Morganstein, 2003). Depressed workers reported a substantially greater number of total health-related lost productive time ($M$ = 5.6 hours/week) than workers without depression ($M$ = 1.5 hours/week). Almost half (48.0%) of the lost productive time among workers who were depressed resulted from MDD, and the majority of their lost productive time was accounted for by decreased job performance. Stewart et al. (2003) found a significant lost productive time cost to employers over the span of 1 week among depressed workers exceeding $44.0 billion/year. Given the high prevalence of nonseasonal depression in the U.S. population (Kessler et al., 2003), the resultant economic burden of lost productive time among workers who are depressed (Stewart et al., 2003), and the moderate to severe symptomatology associated with depression (Kessler et al., 2003), one might expect a similar public health impact for SAD.

A few epidemiological surveys of SAD prevalence have been conducted within specific cities and counties. In Rosen et al.'s (1990) population study of Nashua, New Hampshire adults, 9.7% and 11% met criteria for SAD and S-SAD, respectively. Kasper et al. (1989) and Rosen et al. (1990) found very similar prevalence rates for SAD and S-

SAD in the Central Eastern U.S. In Montgomery County, Maryland, Kasper et al. (1989) found rates of 13.5% for S-SAD and 4.3% for SAD, and Rosen et al. (1990) found prevalence rates of 10.4% for S-SAD and 6.3% for SAD. Both studies showed a negative correlation between seasonal behavior changes and age, suggesting that SAD and S-SAD may occur more frequently in young adults. Within the recurrent depressed population, an estimated 16% of the cases follow a seasonal pattern (Thase, 1986). SAD is more prevalent at Northern latitudes where photoperiod (i.e., the hours of daylight) is shortest in winter. In the Rohan and Sigmon (2000) Northeastern U.S. college student sample, 16% of participants met Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal, Bradt, & Wehr, 1984a) criteria for S-SAD and more than 5% met criteria for SAD.

Epidemiological studies have identified several correlates of SAD, including gender, age, and residing at a high latitude. However, because no prospective, longitudinal studies have been conducted and no odds or risk ratios have been reported, it is unknown whether these represent risk factors for SAD. In a study by Kasper et al. (1989), women comprised 71% of those suffering from SAD, whereas, Terman et al. (1989) found that 68% of SAD cases were women. Rohan and Sigmon (2000) and Rosen et al. (1990) found that SAD occurs about four times more frequently in women than in men. With regard to latitude, research has shown a high correlation (.85) between latitude and SAD prevalence rates (Potkin, Zetin, Stamenkovic, Kripke, & Bunney, 1986). A multi-site U.S. study found a progressive increase in SAD prevalence from the most Southern site (i.e., Sarasota, FL) to the most Northern site (i.e., Nashua, NH; Rosen et al., 1990). Rosen et al. (1990) argued that although this correlation illustrates that

SAD prevalence increases with latitude, latitude per se does not explain all of the variance in SAD prevalence.

A number of other variables correlate with SAD onset including photoperiod, sociocultural factors, genetic influences, and various climatological conditions (i.e., heat and humidity, global radiation, and temperature; Mersch, Niddendorp, Bouhuys, Beersma, & van der Hoofdakker, 1999). In an extensive review of the literature concerning seasonality and latitude, Mersch et al. (1999) argued that the relationship between SAD prevalence and latitude appears inconclusive. This review concluded that SAD prevalence rates in North America were twice as high as prevalence rates in Europe. Significant positive correlations between latitude and SAD prevalence were found only in North American studies (Mersch et al., 1999). These results suggest that latitude may represent only one contributing factor to SAD vulnerability, possibly in a synergistic effect with other environmental factors.

## Mechanisms and Etiology of Seasonal Affective Disorder

### *Biological Models*

The predominant theoretical explanations for SAD adhere to a purely biological perspective, positing causal mechanisms such as phase-delayed circadian rhythms, insufficient amounts of light entering the retina, an abnormal response to changes in duration of melatonin secretion, and decreased receptor specific serotonergic activity (Lee, Blashko, Janzen, Paterson, & Chan, 1997). Through these biologic mechanisms for SAD, manifestation of the disorder is hypothesized to result from reduced exposure to natural sunlight, particularly when photoperiod is short (Lingjærde, Bratlid, Hansen, & Grotestam, 1986; Rosen et al., 1990). However, based on DSM-IV (APA, 1994) criteria

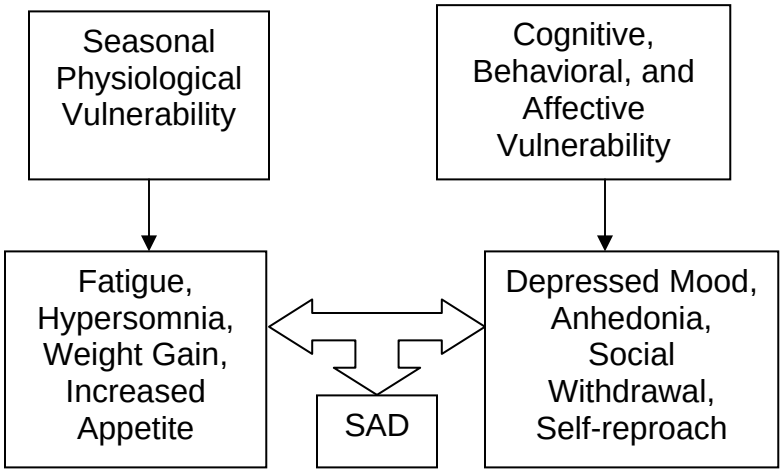
for SAD, an individual must experience not only the physical (vegetative) symptoms, but some combination of cognitive, affective, and physical symptoms (Young, 1999). Given that DSM-IV diagnostic criteria include both cognitive and affective symptoms, Young (1999) argued that a strictly biological model may be insufficient in explaining SAD symptom onset and maintenance. Most studies of SAD from a psychological perspective have been based on the dual-vulnerability model, a preliminary psychological/biological model of SAD.

### *Psychological Models*

***Dual Vulnerability.*** In addition to a biological component, the dual vulnerability model incorporated a second (i.e., psychological) component to explain the development of SAD (Young, Watel, Lahmeyer, & Eastman, 1991). According to the model, individuals with seasonal depression exhibit two separate vulnerabilities. First, they may have a preponderance for experiencing reverse vegetative symptoms (e.g., increased sleep, increased appetite and carbohydrate craving, and increased fatigue) in conjunction with the changing seasons, which constitutes a physiologic "vulnerability." Second, in response to the physiological symptoms, cognitive and affective disturbances ensue, which indicates activation of the psychological component of the "dual" vulnerability (See Figure 1).

***Figure 1.***

**Young's Dual Vulnerability Model.**




Source: Young et al. (1991).

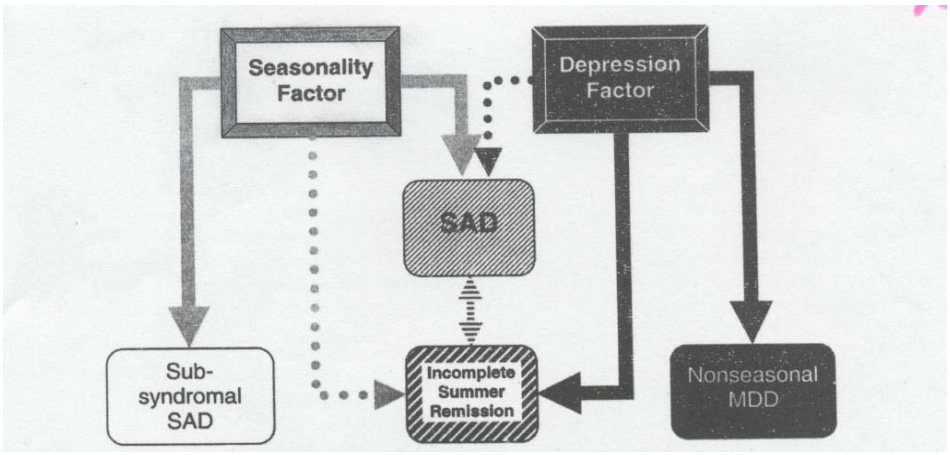
The Young (1999) model, however, may be too simplistic and reductionistic in its explanation of the factors that contribute to the onset of SAD. This model assumes that the reverse vegetative symptoms occur first, followed by cognitive and affective expressions of depression, but there are no prospective studies to confirm this temporal course. In reality, there is no evidence that cognitive, affective, and behavioral symptoms are linked to a psychological vulnerability, whereas vegetative symptoms are tied to a physiological vulnerability. This linear explanation may not accurately reflect the sequence of events that contribute to SAD development. It is likely that these two "vulnerabilities" are intertwined in some complex way, making it difficult to tease apart whether one vulnerability factor precedes another. Rather, it may be more accurate to describe the physiologic and psychologic vulnerabilities as bio-behavioral correlates of depression.

Young et al.'s (1991) dual vulnerability model has been extended to a spectrum of disease model. In a purely conceptual model, Lam, Tam, Yatham, Shiah, and Zis (2001) proposed that there are two basic "loading factors" that may contribute to whether depressive symptoms are expressed in a purely seasonal, nonseasonal, or mixed pattern of recurrence. These two factors, the seasonality factor and the depression factor, may differ in strength across diagnostic categories (i.e., SAD, S-SAD, and MDD). In S-SAD, there is very little or no loading on the depression factor and primary loading on the seasonality factor. This combination is expressed through the individual's experience of the reverse vegetative symptoms with no concomitant experience of the affective and cognitive symptoms essential for a diagnosis of a nonseasonal Major Depressive Episode. An individual with primary loading on the depression factor with minimal loading on the seasonality factor would most likely experience "pure" MDD, the polar opposite of SAD on the expression spectrum, resulting in experience of more "typical" nonseasonal depression symptoms including loss of appetite and insomnia.

Along the middle of the continuum lie SAD and SAD with incomplete summer remission, representing intermediate loadings on the depression and seasonality factors. In the case of primary loading on the seasonality factor, the most likely expression is that of diagnosable SAD and the experience of complete remission of symptoms in spring and summer. Alternatively, higher loading on the depression factor, as compared to the seasonality factor, results in expression of a winter Major Depressive Episode without full remission of symptoms coinciding with the arrival of spring and summer (See Figure 2).

***Figure 2.***

**Spectrum of Disease Model.**




Source: Lam et al. (2001).

Further, Lam et al. (2001) hypothesized that a "trait-like" vulnerability to seasonality becomes activated by environmental cues (e.g., photoperiod) to contribute to a high loading on the seasonality factor, whereas a "trait-like" vulnerability to depression, compounded by external consequences (e.g., life stressors) results in a high loading on the depressive factor. This "trait-like" vulnerability represents a predisposition to recurring bouts of depression that may have developed because of a conditioning history of seasonal cues and the onset of depression symptomatology.

In addition to work aimed at developing integrative biological and psychological models to explain the etiology of SAD, other researchers have applied psychological models of nonseasonal depression to SAD. Most of this work has focused on cognitive factors that may contribute to the development and/or maintenance of a SAD episode. Cognitive factors borrowed from Beck's model, known as automatic negative thoughts, dysfunctional attitudes, and negative core schemas have been hypothesized to directly

contribute to onset of a SAD episode, or to exacerbate the severity and duration of a current seasonal depressive episode (Hodges & Marks, 1998; Rohan, Sigmon, & Dorhofer, 2003).

### *Cognitive Models*

***Beck's Cognitive Model of Depression.*** According to the cognitive model of depression (Beck, 1967; 1976), cognitive schemas represent the highest level of information processing and are comprised of attitudes or assumptions that are both global and stable. Schemas constitute the core beliefs one holds about oneself, the world, and the future, presumably learned during childhood. Individuals develop both positive and negative schemas, but the negative ones generally remain inactive when mood is nondepressed. Persistent and, oftentimes, pervasive negative schemas are activated under conditions of stress and contribute to depression onset and maintenance (Beck, Rush, Shaw, & Emery, 1979).

Once negative schemas are activated, individuals experience recurring cognitions with distorted, negative connotations or content. These unrealistic, negative thought processes include dysfunctional attitudes (i.e., higher level, maladaptive rules or assumptions that guide behavior), and, on a conscious level, automatic thoughts (i.e., spontaneous mental activity in reaction to specific events). When negative schemas are operative, automatic thoughts are negative and drive a negative emotional reaction to an antecedent event.

In an application of Beck's cognitive model of depression to SAD, dysfunctional attitudes and automatic thoughts have been examined in comparisons of seasonal and nonseasonal depression, and in comparison between individuals with SAD and

nondepressed controls. One preliminary study found that a group of women with a history of SAD endorsed more automatic negative thoughts, regardless of the season (i.e., winter, summer, and fall) relative to nondepressed controls (Rohan et al., 2003). Women with SAD history also reported an increased frequency of negative automatic thoughts in winter as compared to summer. A second study (Hodges & Marks, 1998) found no differences in automatic negative thought frequency between currently depressed individuals with SAD and nonseasonal depression, but both groups were elevated above nondepressed controls.

In a longitudinal comparison of college women with S-SAD and a group of nonseasonal, nondepressed controls, Rohan et al. (2004) found that S-SAD individuals endorsed more automatic negative thoughts during the winter and during a nonwinter month (i.e., either spring or fall) when compared to the control group with more frequent automatic negative thoughts in winter than in nonwinter. Because this pattern of negativistic thinking in SAD and S-SAD occurred in both winter and nonwinter months, negative automatic thoughts may represent a "trait-like" cognitive phenomenon that is pervasive, across the seasons. In contrast, in nonseasonal MDD, negative automatic thoughts appear to be more "state-like" because they wax and wane with depressive symptomatology (Hollon, Kendall, & Lumry, 1986). One potential limitation of these studies (Rohan et al., 2003; 2004) is that the SAD and S-SAD samples were not directly compared in the same study.

Regarding dysfunctional attitudes, Hodges and Marks (1998) found that individuals with SAD evidenced similar dysfunctional attitudes to individuals with nonseasonal depression, and that both groups differed from nondepressed controls. In

contrast, in a comparison of women with a history of diagnosed clinical SAD and a nondepressed control sample, Rohan et al. (2003) found no significant group differences in dysfunctional attitudes. Only seasonal differences in dysfunctional attitudes emerged. Women with a history of SAD reported more dysfunctional attitudes in fall than summer, and the control group endorsed greater dysfunctional attitudes in fall than winter.

A study by Levitan, Rector, and Bagby (1998) suggested a parallel negative attributional style between individuals with seasonal and nonseasonal depression. A negative attributional style refers to the tendency to ascribe global and stable attributions to negative situations. This research is consistent with the finding that negative automatic thoughts and dysfunctional attitudes are comparable in seasonal and nonseasonal depressive episodes (Hodges & Marks, 1998). These studies examining automatic thoughts, dysfunctional attitudes, and negative attributional style provide evidence that, similar to nonseasonal depression, negative cognitive-processing biases are correlates of SAD and S-SAD.

***Response Styles Theory.*** Another cognitive model of depression that may help to explain SAD onset and maintenance is response styles theory (Nolen-Hoeksema, 1987). This theory hypothesizes that rumination, or a deliberate, repetitive focus on one's reasons for depressed mood as well as the anticipated outcomes, constitutes a "trait-like" vulnerability to development of more severe, prolonged depressive symptoms. Rumination has been shown to promote a negative attributional style as described in Beck's model of depression (Lyubomirsky, Caldwell, & Nolen-Hoeksema, 1998).

There is substantial evidence in the nonseasonal depression literature that increased rumination prolongs and intensifies periods of mood disturbance (Nolen-

Hoeksema & Morrow, 1991; Nolen-Hoeksema, Morrow, & Frederickson, 1993), and contributes to the development of MDD (Just & Alloy, 1997). Moreover, the other response style, distraction, may possess anti-depressant properties. Lyubomirsky et al. (1998) found that when mildly depressed individuals engaged in distracting behaviors (i.e., thinking about something other than their depressed mood), their symptom duration and intensity decreased.

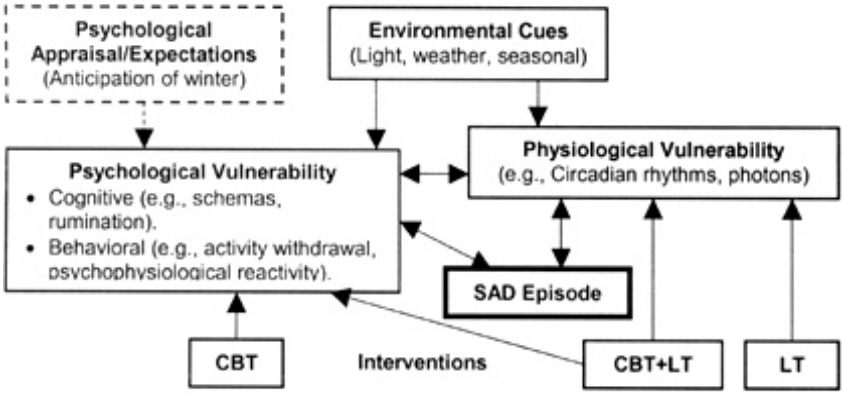
Two studies that have applied the response styles theory to SAD suggest that rumination may constitute a cognitive vulnerability for SAD onset. Rohan et al. (2003) found that individuals with a history of SAD tended to ruminate (i.e., focus on the causes and consequences of their depression) at greater frequency than controls as demonstrated by higher scores on the Response Styles Questionnaire (RSQ) – Rumination subscale (Nolen-Hoeksema & Morrow, 1991). Importantly, participants' fall rumination frequency significantly predicted the severity of their seasonal symptoms during the subsequent winter, over and above fall level of depressive symptoms. In another study, Azam and Young (2001) demonstrated that frequency of self-recorded ruminative behaviors in the fall predicted depressive symptom severity the following winter. Rumination, therefore, may represent a cognitive vulnerability to SAD onset and may increase the intensity and duration of season-related symptoms.

***Integrative Cognitive-Behavioral Model.*** In an effort to extend the dual vulnerability model and to explicate the content of the psychological vulnerability, Rohan (2002) formulated an integrative cognitive-behavioral model of SAD. Similar to dual-vulnerability, the model hypothesizes that psychological and physiological processes are

interrelated in a complex system to produce depressive symptom onset and maintenance in SAD (See Figure 3).

***Figure 3.***

**Integrative, Cognitive-Behavioral Model.**




Source: Rohan (2002).

Unlike dual-vulnerability, Rohan's model posits that the psychological vulnerability consists of negative cognitive processes (e.g., negative automatic thoughts, dysfunctional attitudes, schemas, and rumination) and behavioral responses (e.g., withdrawal from reinforcing activities, psychophysiological reactivity). In this model, an individual's psychological appraisal of, or expectations for depressive symptoms coinciding with the onset of the fall and winter seasons, may be sufficient provocation to activate one's psychological vulnerability to SAD. In addition, the model illustrates that numerous environmental cues (e.g., decreased photoperiod, temperature changes, and seasonal cues such as fall foliage) may activate both the psychological and physiological (e.g., phase-delayed circadian rhythms, increased melatonin secretion) vulnerabilities to SAD, thus contributing to SAD episode onset. Once the system is activated, the model proposes that the psychological and physiological vulnerabilities act in a reverberating

circuit to maintain the episode until the environmental cues change (e.g., photoperiod lengthens, temperature rises, and grass/trees turn green).

As stated above, psychophysiological reactivity is listed as a component of the psychological vulnerability to SAD. Specifically, Rohan's model proposes that individuals with SAD demonstrate heightened psychophysiological responses to environmental stimuli that have been paired with a SAD episode. Consequently, the heightened responses may occur irrespective of current season in a more "trait-like" pattern, but are expected to be attenuated during spring and summer. Cues associated with the winter season (e.g., stark trees) and low light availability (e.g., dreary skies) are thought to be especially salient. Examination of psychophysiological reactivity to both light- and season-relevant stimuli in SAD are needed to test this component of the model. This is important because the model specifically incorporates both environmental cues and the anticipation of depressive symptoms with the onset of winter as essential to the development of a SAD episode. Before reviewing what is known about psychophysiological reactions in SAD, emotional responses to light- and season-relevant stimuli will be discussed.

## Emotional Reactivity to Light- and Season-Related Stimuli

### *Cognitive Sensitivity to Light*

As previously reviewed, light availability, primarily photoperiod, may relate to SAD prevalence and onset because as latitude increases, SAD prevalence rates also tend to increase (Rosen et al., 1990). Statistical correlations between latitude and SAD, S-SAD, and combined prevalence rates indicate a strong positive relationship between latitude and prevalence, at least in the United States (Mersch et al., 1999; Rosen et al.,

1990). A few researchers have proposed psychological mechanisms to link light availability to SAD onset.

Bouhuys, Meesters, Jansen, and Bloem (1994) hypothesized that a cognitive sensitivity to light-relevant stimuli may be related to the onset and maintenance of SAD; thus, a decrease in the photoperiod in winter may initiate a "negative processing bias." Using an experimenter-developed schematic (i.e., line-drawn) faces task, Bouhuys et al. (1994) found that participants with a history of SAD demonstrated an increased cognitive sensitivity to symbolic light, making more negative attributions to schematic faces when embedded in dark versus light backgrounds relative to nondepressed controls. Further, larger differences in rated "activation" between the dark and light backgrounds were associated with an earlier onset of depression the following winter.

***Sensitivity Hypothesis***

In an examination of emotional reactivity to light-relevant stimuli, Rohan et al. (2003) extended Lewinsohn, Hoberman, Teri, and Hautzinger's (1985) sensitivity to aversive events hypothesis to SAD. That is, as in depression, individuals with SAD may be especially sensitive to aversive events. However, what is most aversive to nonseasonally depressed individuals (e.g., interpersonal rejection) may be different than what individuals with SAD perceive as most aversive (e.g., low light availability). As a task to test this hypothesis, participants viewed three blocks of slides containing outdoor scenes (six slides per condition) on a computer. Each block of slides depicted a different light intensity: bright light (e.g., sunny), low light (e.g., dark, dreary), and ambiguous light (e.g., sky not visible in slides).

Rohan et al. (2003) found that women with a history of SAD experienced improvement of mood in reaction to exposure to bright light stimuli relative to nondepressed controls; thus, exposure to a bright light stimulus appeared to act as an indirect mood enhancer in SAD. Rohan et al. (2003) found a greater exacerbation of baseline depressive mood state after exposure to low light slides versus ambiguous light or bright light slides among women with SAD history. These results support previous findings indicating that individuals with SAD demonstrate reactivity to light-relevant stimuli. Perhaps one explanation for this pattern of findings is that women with a history of SAD may be demonstrating a classically-conditioned emotional response to stimuli associated with a depressive episode, as evidenced by their increased self-reported depressive mood state following exposure to low light stimuli.

In an S-SAD sample, Rohan et al. (2004) found that presentation of bright light stimuli resulted in a greater improvement of self-reported affective state than controls. S-SAD participants also reported an exacerbation of baseline depressed mood following presentation of low light stimuli relative to bright light scenes. Results of these two studies using the same task (Rohan et al., 2003; 2004) suggest that individuals who develop SAD and S-SAD demonstrate heightened emotional reactivity to light-related stimuli as compared to nondepressed controls.

In addition to studying reactivity to light-relevant stimuli, researchers have investigated emotional reactivity to seasonal stimuli in SAD. Sigmon et al. (2002) used a video task involving winter and summer images while measuring skin conductance response as a psychophysiological marker. These researchers compared participants meeting DSM-IV criteria for Major Depressive Disorder, Recurrent, with Seasonal

Pattern Specifier (SAD), individuals diagnosed with nonseasonal Major Depressive Disorder (MDD), individuals with subsyndromal SAD (S-SAD), individuals with a SAD history who were not currently in a depressive episode (SAD-HX), and control participants with no history of depression and a concurrent low seasonality score as assessed with the Seasonal Pattern Assessment Questionnaire (SPAQ; Rosenthal et al., 1984a).

To examine changes in subjective report of depressed mood using the Depression - Dejection subscale of the Profile of Mood States questionnaire (POMS; McNair, Lorr, & Droppleman, 1971), change scores were computed from the time participants watched a video depicting winter scenes to the time they observed summer scenes (i.e., winter – summer POMS; Sigmon et al., 2002). This computation of change scores was necessary given that there were significant baseline differences in depressed mood across groups. Results showed that participants in the SAD and MDD groups experienced greater changes in depressed mood (in the direction of more depressed mood to winter than to summer scenes) than individuals in the SAD-HX and control groups. SAD participants were not significantly different from the MDD and S-SAD groups. The S-SAD group reported greater depressed mood change than controls, and there were no differences between the SAD-HX group and controls. These results suggest increased emotional reactivity to winter stimuli among individuals with SAD, S-SAD, and MDD.

## Psychophysiological Reactions and SAD

"Psychophysiology is the study of relations between psychological manipulations and resulting physiological responses, measured in the living organism, to promote understanding of the relation between mental and bodily processes" (Andreassi, 1995).

One method of assessing psychophysiologic response is by measuring skin conductance, or the electrical activity of the skin (Stern, Ray, & Quigley, 2001). Skin conductance measurement is important because the degree of sympathetic nervous system activation causes the eccrine in sebaceous sweat glands to produce and secrete varying amounts of sweat. The amount of sweat produced in a sweat gland is a measure of sympathetic activity; the lower the skin resistance[1] the higher the skin conductance.[2] Therefore, the psychophysiological measurement of skin conductance demonstrates the degree of sensitivity to a specific stimulus, as evidenced by increased sweat production.

***Preliminary Findings***

According to Lewinsohn's integrated model of depression, individuals are vulnerable to depression if they develop a hypersensitive response to aversive stimuli (Lewinsohn et al., 1985). This vulnerability could reveal itself as either passive avoidant learning and/or through a heightened physiologic reaction to unpleasant stimuli (Sigmon, Hotovy, & Trask, 1996; Sigmon & Nelson-Gray, 1992). In the nonseasonal depression literature, currently depressed, previously depressed, and dysphoric individuals exhibit greater psychophysiological responding (e.g., skin conductance) to unpleasant stimuli when compared to nondepressed controls (Sigmon & Nelson-Gray, 1992; Sigmon et al., 1996). Using Lewinsohn et al.'s (1985) theoretical model as a basis, Rohan et al. (2003) theorized that a clinical SAD population would also demonstrate this heightened reactivity to unpleasant stimuli.

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[1] Skin resistance – exosomatic electrodermal activity measured as resistance of the skin to an imposed current, directly measured by a constant current technique (Venables & Christie, 1980).

[2] Skin conductance – exosomatic electrodermal activity measured as conductance of the skin to impressed potential, directly measured by constant voltage techniques (Venables & Christie, 1980). Skin conductance is the reciprocal value of skin resistance.

Assuming that exposure to low light stimuli is unpleasant to a seasonally-depressed sample, Rohan et al. (2003) hypothesized that SAD vulnerable individuals may demonstrate increased skin conductance reactivity to low light stimuli. Two preliminary studies have tested this hypothesis. First, Rohan et al. (2004) found that during the winter months, women with S-SAD demonstrated higher skin conductance response (SCR) magnitude than nondepressed, nonseasonal controls when presented with low light stimuli. Regardless of the season, S-SAD women also demonstrated greater SCR magnitude when presented with low light scenes relative to bright light or ambiguous light scenes. A second study (Rohan et al., 2003), however, found no statistically significant differences in SCR magnitude between SAD individuals and nondepressed controls across the seasons (e.g., fall, winter, and summer) or stimuli type (e.g., bright light, low light, and ambiguous light).

It is possible that no significant group differences were found in this study because the majority of participants were middle-aged women, and skin conductance negatively correlates with age (e.g., older individuals emit reduced skin conductance responses; Anderson & McNeilly, 1991). Therefore, skin conductance may not have been a sensitive enough psychophysiological measure to detect reactivity in the SAD sample. There are several other possible explanations for the null findings in Rohan et al. (2003). First, the sample size was relatively small (i.e., 18 women with a history of SAD and 20 matched-controls) and comparisons could not be made between those individuals with a history of SAD who had experienced a current Major Depressive Episode and those who did not. In addition, the nonsignificant findings could be due to individual differences.

Given that Rohan's (2002) model of SAD proposes increased reactivity to unpleasant environmental stimuli representing winter cues, winter-relevant stimuli may also elicit psychophysiological responses. Sigmon and colleagues (2002) demonstrated that participants with any type of significant seasonal history or current seasonal depression (i.e., SAD, S-SAD, and SAD-HX) exhibited more significant skin conductance responses (SCRs) as well as increased SCR magnitude to a video of the state of Maine (i.e., seasonal, environmental) depicting winter scenes when compared with the MDD and control groups. There were no significant differences between the control group and those with MDD on SCRs or SCR magnitude. Interestingly, the groups did not differ in skin conductance reactions to the summer video scenes. The Sigmon et al. (2002) study is the first to employ a measure of psychophysiological reactivity (i.e., skin conductance) to season-related stimuli in an attempt to determine differential response in nonseasonal depression versus varying degrees of SAD. These results have significant implications for the current study, which will examine psychophysiological response to both light- and season-relevant stimuli simultaneously.

One substantial weakness of these preliminary studies is their exclusive focus on skin conductance. As stated previously, skin conductance reflects, to a certain extent, general sympathetic arousal. Thus, increased skin conductance reactivity to stimuli suggests that those stimuli are meaningful. Skin conductance reactions, however, do not provide information about whether the stimuli evoke mood-specific reactions.

## Psychophysiological Reactivity and the Menstrual Cycle

It is well known that many women experience fluctuations in mood and behavior, and physical states across the various stages of their menstrual cycle (Moos et al., 1969).

The reported changes can include vacillations in irritability, depressed mood, energy level, and physiological sensations (Logue & Moos, 1986). Importantly, most studies have associated increased physical and mental symptoms with the premenstrual, or late phase luteal, phase of the menstrual cycle (Altman, Knowles, & Bull, 1965; Gallant, Hamilton, Popeil, Morokoff, & Chakraborty, 1991). Research has also shown that menstrual cycle phase can represent a confound in psychophysiological studies. One study compared differences in baseline resting levels of reactivity between women in the follicular phase as compared to women in the luteal phase. Results demonstrated differences in physiological responding in heart rate, pulse transit time, and EEG alpha wave activity during completion of tasks, but no differences in reactivity (Kaplan, Whitsett, & Robinson, 1990). Another study using a sample of 67 female undergraduates found that negative moods peaked during the luteal phase of the menstrual cycle (Rossi & Rossi, 1977), which is consistent with results from a different study that demonstrated that 50% of the healthy young women in the sample (mean age of 22 years) experienced the most profound depressed mood while in the luteal phase of their menstrual cycle (May, 1976).

Studies have also shown that women with high levels of anxiety sensitivity, particularly women with panic disorder, may experience the largest increases in psychological distress as well as heightened SCR psychophysiological reactivity during the premenstrual phase relative to control women. Both high anxiety sensitivity women and women with panic disorder reported more severe menstrual symptoms and exhibited increased psychophysiological responses as measured by SCR frequency and SCR magnitude, to anxiety-relevant stimuli during the premenstrual phase relative to low

anxiety sensitivity control women (Sigmon, Dorhofer, Rohan, Hotovy, Boulard, & Fink, 2000; Sigmon, Fink, Rohan, & Hotovy, 1996). Therefore, it is reasonable to assume that women with seasonal depression may exhibit greater psychophysiological reactivity, depending upon their current phase of the menstrual cycle.

Given that the present study is concerned with emotion-specific reactivity, menstrual cycle-related changes in physical and affective states may affect female participants' psychophysiological response patterns, particularly if they are in the premenstrual phase during the assessment. Therefore, it is important for the present study to assess current menstrual cycle phase in female participants to determine if there are differences in emotion-specific reactivity (i.e., EMG, SCR) based on menstrual cycle phase. For this reason, we are collecting descriptive information during the assessment concerning each female participant's menstrual cycle phase (See Appendix A).

## Surface Facial EMG and Depression

### *Electromyography*

Electromyography (EMG) is the measurement of muscular electrical activity produced when muscle fibers fire. More specifically, facial EMG can refer to the patterning of facial muscle activity as expressed during emotional reactions (Andreassi, 1995). Surface EMG equipment records an algebraic sum of the number of depolarizations that occur when groups of motor units (i.e., muscle groups of interest) are activated. In contrast to skin conductance, which measures general arousal, EMG represents a more precise measure to differentiate between positive or negative emotions that has been widely studied in nonseasonal depression and could be extended to SAD. Given that sensitivity to environmental cues associated with season and light availability

is hypothesized as a component of the psychological vulnerability to SAD (Rohan, 2002), EMG represents a more useful modality for measuring emotion-specific psychophysiological reactivity.

The results of Rohan et al. (2004) and Sigmon et al. (2002) suggest that there is an underlying psychophysiologic reactivity to light- and season-relevant stimuli in seasonality populations. The emotional specificity of this reactivity may be more effectively captured through the highly sensitive, surface facial EMG measurement, given the prevailing evidence that facial EMG is an especially sensitive measure of emotion in clinical, nonseasonal depression (Schwartz et al., 1976a).

Given the increased self-reported depressed mood in SAD upon exposure to both low light and winter stimuli and the improvement in mood subsequent to bright light stimuli, there is a need to combine the two conditions to examine any interactions or synergistic effects that individuals with SAD are responding to. No study to date has simultaneously explored both light and seasonal stimuli in SAD. It may not be simply light or season alone that individuals are reacting to, but rather an interaction between the two that serves to elicit a negative affectively-charged response. Through muscle patterning and expression of the human face, surface facial EMG constitutes a potentially ideal means of examining both depressed emotional state in reaction to low light, winter stimuli and happy affect during bright light, summer stimuli.

### *Facial Muscles and Specific Emotions*

The earliest and perhaps one of the most important connections between facial expressions and emotions was introduced by Darwin (1872). More than a century ago, Darwin posited that humans are physiologically hard-wired and congruent in their

expression of emotion, a concept that has received extensive empirical support, to date. Darwin (1965; 1872) also was the first to introduce the concept of examining facial affective expressions in reaction to photographic stimuli (e.g., pictures of his own child pouting and laughing).

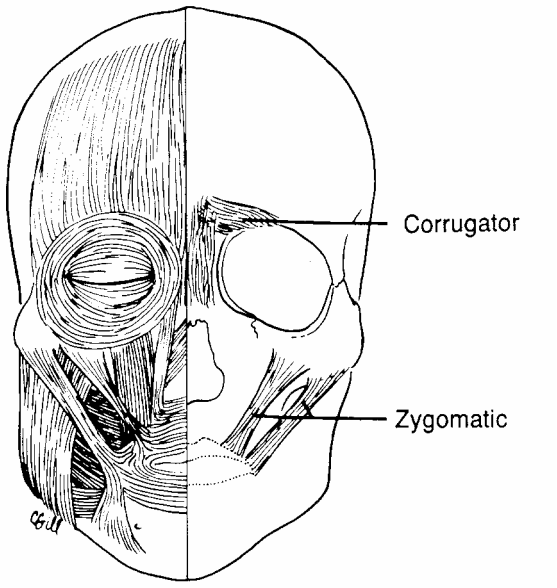
Eckman, Friesen, and Ellsworth (1972) and Izard (1971), in a cross-cultural comparison of facial expression, established that there are six recognized facial expressions generally categorized as specific emotions including sadness, happiness, anger, fear, surprise, and disgust. Observations of overt facial expression and self-report measures have been used to capture these emotions. However, these gross facial movements are not well suited for measurement of small muscular activations occurring in the facial region, especially those that occur in a matter of milliseconds. EMG is a more effective measure of affective response. To demonstrate this phenomenon, Cacioppo, Martzke, Petty, and Tassinary (1988) recorded EMG response in the brow region (i.e., corrugator muscle) in a group of participants that were interviewed and asked to describe themselves. While being interviewed, facial expressions emitted were video-taped. Following the interview, individuals were asked to describe what they were feeling at specific points during the interview when significant corrugator activity was recorded. Results suggested that EMG is a reliable psychophysiological measure of subtle facial muscle activity, not detectible through direct observation, which represents a correlate of emotion.

There have been numerous studies showing that facial muscle movement can be tied to the experiencing of specific emotions. Tassinary, Cacioppo, and Geen (1989) provided psychometric support that particular recording sites in the vicinity of specific

facial muscle groups have higher sensitivity to detect muscle-specific reactivity and are, therefore, valid indices bridging activation of muscle electrical potentials to facial movement. The first muscle group, the zygomaticus major, located near the side of the mouth and extending to the upper cheek, is mainly responsible for producing a smile (Carney, Hong, O'Connell, & Amado, 1981). The other prominent muscle group is the corrugator supercilii located on either side of the bridge of the nose, near the center of the eyebrow, and is thought to primarily control the production of a frown, including the act of brow-knitting (Carney et al., 1981). These regions, therefore, represent optimal placement of EMG electrodes for effective EMG measurement of facial emotional responses (See Figure 4).

***Figure 4.***

**Anatomic Illustration of Facial Musculature.**




Source: Greden et al. (1986).

## Facial EMG and Nonseasonal Depression Studies

### *Facial Patterning in Depression*

Facial muscle movement and patterning is relevant to understanding emotional responses in both seasonal and nonseasonal depression. In contrast to the small SAD literature, researchers have been examining EMG responses in nonseasonal depression for nearly 3 decades. Schwartz et al. (1976a) conducted some of the first studies that examined facial expression as it relates to emotion in clinical depression using EMG. Results from this study showed that facial EMG can reliably differentiate between happy and sad imagery across nondepressed and clinically-depressed populations, with the corrugator and zygomatic muscles proving especially sensitive for measuring sad and happy affective states, respectively. First, this research demonstrated that EMG measurements could detect subtle "covert" facial expressions (i.e., chronic facial muscle tension, not detectible by the naked eye), which is important given that depression is often accompanied by chronic patterns of muscle tension in the head (e.g., forehead, jaw-tongue; Lundervold, 1952; Plutchik, 1954; Whatmore & Ellis, 1959), thereby demonstrating the possible predictive utility of surface facial EMG.

Second, differences also were found between EMG patterning when participants were instructed to close their eyes and "re-experience" the feelings associated with mental generation of personally-relevant happy or sad imagery. Facial muscle activity in this condition was greater than when participants were told to simply focus on the imagery, without self-inducing a matching affective state (i.e., happy or sad). Relative to nondepressed participants, the depressed group demonstrated greater attenuation of facial EMG patterns when asked to imagine a happy image than when asked to self-regulate an

actual re-experiencing of happy imagery. Thus, when specifically asked to do so (i.e., the "feel" condition), depressed individuals could self-generate happy affect similar to nondepressed individuals. However, when just thinking about happy imagery, they could not spontaneously generate a muscle pattern consistent with happy emotions as nondepressed individuals do. This study indicated that measurement of facial patterning of muscle activation could be used in assessing both nondepressed and clinical mood states.

In another early study (Schwartz, Fair, Salt, Mandel, & Klerman, 1976b), clear differences were found in facial EMG activity between depressed and nondepressed participants. Comparisons were made across three separate emotions (i.e., happy, sad, and typical day) in a sample of 24 females, 12 of whom were classified as "normal" and 12 volunteers classified as depressed. Results were calculated as change scores from baseline EMG activity, and averaged over all trials. For the total sample, happiness was generally associated with decreased corrugator activity and sadness was generally associated with increased corrugator activity. Despite similar sadness and anger response patterns among the groups, depressed participants evidenced an attenuated pattern of responding in the happiness condition compared to the normal controls. When imagining a typical day, depressed participants demonstrated a "sad" pattern (i.e., an increase in lateral and medial frontalis activity, a muscle group across the forehead involved in the act of frowning), and nondepressed participants exhibited a "happy" pattern (i.e., a decrease in corrugator activity). Additionally, when self-evaluating affective mood states, each group reported results paralleling those measured with facial EMG (e.g., nondepressed participants reported greater feelings of happiness in the "happy"

condition, and less sadness in the "typical day" condition relative to depressed participants). Both of these studies by Schwartz and colleagues (1976a; 1976b) demonstrate that EMG is a sensitive measure of emotions in normal and clinical samples.

Corrugator and zygomatic surface facial EMG also has been applied to other types of stimuli, specifically, slides of social scenes (e.g., facial expressions indicative of specific emotions) and scenery (e.g., mountains; Cacioppo et al., 1992; Cacioppo, Petty, Losch, & Kim, 1986). Normal female participants were exposed to equal numbers of pleasantness-matched faces and scenes described as pleasant (e.g., happy facial expression, a mountain cliff), unpleasant (e.g., angry facial expression, bruised torso), or neutral (e.g., unexpressive facial photograph, ocean beach, polluted roadway). Participants were instructed to exaggerate their reaction to the stimuli, to inhibit their affective reactivity, or were given no verbal guidance. Results showed that corrugator activity was highest when viewing unpleasant scenes and lowest when viewing pleasant scenes. Overall, facial EMG activity was highest in the "exaggerate" condition and lowest in the "inhibit" condition. EMG response did not differ when viewing "pleasantness-matched" social or environmental scenes. This study demonstrates that surface facial EMG is a valid psychophysiological marker of emotional response to affect-laden stimuli, regardless of whether participants demonstrate an overt change in facial expression.

Greden et al. (1986) showed that nondepressed controls produced greater facial EMG patterning responses than those diagnosed with MDD (endogenous type) when asked to imagine various imagery states (e.g., typical day, sad, and happy). This suggests that individuals with depression may be more blunted in reaction to different types of

imagery relative to nondepressed individuals. Participants with endogenous depression were found to have significantly higher absolute values in corrugator muscles in both sad (e.g., a personally-relevant circumstance that made the participant feel sad) and happy (e.g., a personally-salient scenario that evoked happy emotions) imagery states when compared to those with non-endogenous depression. These results suggest that participants with endogenous depression appear to have a more reactive corrugator muscle regardless of the imagery state (i.e., happy or sad). Consequently, those with endogenous depression may have an overactive "grief" muscle group, irrespective of whether happy or sad imagery is presented. In summary, there appear to be clear differences in facial muscle patterning for endogenously depressed versus nondepressed individuals.

Oliveau and Willmuth (1979) further supported the notion that facial EMG responses differed according to the context of affect-producing imagery. In this case, measurable changes from baseline corrugator activity were recorded in both depressed and nondepressed participants when told to recall and "picture in their mind" some sad circumstance (e.g., self-regulation of a vividly recalled sad circumstance), however the differences between the groups were not statistically significant. Additionally, there were no significant changes from corrugator baseline EMG when participants were asked to imagine happy circumstances, regardless of group. Further, only the depressed group evidenced significant EMG response change from baseline in the typical day condition. In support of Schwartz and colleagues (1976a; 1976b), significant changes from baseline were recorded when comparing happy and sad imagery states. However, Oliveau and Willmuth (1979) were unable to differentiate between the sad and typical day imagery

states. Contrary to other research, all of the significant changes from baseline found in this study tended to demonstrate increased corrugator EMG activity during happy, sad, and typical day imagery states.

In a more recent study of surface facial EMG among college undergraduate students who were dysphoric (not meeting diagnostic criteria for depression), reactivity to pictures depicting different types of facial expressions drawn from the International Affective Picture System (IAPS; Lang, Bradley, & Cuthbert, 1999) was measured and compared to a group of non-dysphoric students (Sloan, Bradley, Dimoulas, & Lang, 2002). Both corrugator and zygomatic muscle activity were recorded and results indicated that when viewing pictures that included people with unhappy expressions, both groups of participants showed a resultant increase in corrugator muscle group activity (e.g., brow-pursing). However, when examining photographs depicting individuals with happy facial expressions, individuals who were dysphoric did not demonstrate analogous increases in zygomatic muscle activity (e.g., smile response) as did those in the non-dysphoric control group. Surprisingly, dysphoric college students exhibited an increase in corrugator muscle group activity when viewing pictures with happy facial expressions, a result that is consistent with Greden et al. (1986).

The Sloan et al. (2002) results could be explained if individuals who are dysphoric have an overactive “grief” muscle, or perhaps evidence a general pattern of increased corrugator activity (e.g., frowning), regardless of whether presented with photographs or imagery containing positive or negative content (Greden et al., 1986). Thus, it is possible that dysphoric or depressed individuals are not able to exhibit appropriate facial reactivity when presented with happy stimuli, which are inconsistent

with their current mood state. Alternatively, it is possible that there may be a cognitive explanation for this pattern of findings. Perhaps the increased corrugator activity in dysphoric populations under conditions incorporating happy facial expressions is reflective of an awareness of the discrepancy between the happy imagery and their own negative mood state. For example, under a happy facial expression condition, dysphoric individuals may be thinking thoughts such as, "Those people are happy and I'll never be happy" or "I don't have anything in life to be happy about," thus producing increased corrugator activity consistent with their inner experience of dysphoria.

Although there are exceptions (e.g., Greden et al., 1986), this body of research (4 of 6 studies) generally demonstrates that increased corrugator activity is associated with experiencing unhappy, unpleasant, or negative affective imagery in depressed, nondepressed, dysphoric and nondysphoric individuals. Table 1 summarizes the studies that support this conclusion. In addition, decreased corrugator muscle reactivity has been found when viewing happy stimuli in nondepressed undergraduate samples (Cacioppo et al., 1992). Of the 4 studies that considered zygomatic activity, Cacioppo et al. (1992) and Schwartz et al. (1976a) demonstrated increased zygomatic muscle reactivity when exposed to happy, pleasant, or positive affective imagery in both depressed and nondepressed samples.

In contrast, Sloan et al. (2002) found decreases in zygomatic activity in dysphoric students upon exposure to happy facial expressions. Perhaps one reason for the difference in results across studies is because of the authors' assumptions that the stimuli are happy or sad to their various participant groups. If future studies validated the stimuli on the general population (e.g., the IAPS), the perceptions of nondepressed individuals'

may be different than those perceptions of clinically-depressed individuals concerning what constitutes happy or sad imagery. There also appears to be a qualitative difference between surface facial EMG reactivity when comparing clinically-depressed patients with dysphoric college students. Another factor that may help explain these differences in results are the differing methodologies. Because some studies used personally-relevant happy and sad imagery as stimuli (e.g., Greden et al., 1986), it is possible that participants are able to emit increased zygomatic activity (i.e., smile response) when imagining their own happy memories, as compared to viewing stimuli that is assumed to be happy.

## Individual Differences in Imagery Ability

Studies have identified significant individual differences in imagery ability (McKelvie, 1995; Miller, Levin, Kozak, Cook, McLean, & Lang, 1987; Sutherland, Harrell, & Isaac, 1987). In one study, affective and action-oriented instructions elicited greater levels of physiological responding (e.g., heart rate, skin conductance level, and respiration) for individuals classified as "good imagers," compared to those classified as "poor imagers," especially after receiving active imagery response training (Miller et al., 1987). In this small group training session (approximately three individuals per group), participants were first taught progressive muscle relaxation to reduce both between- and within-subjects variability on basal physiological levels. The imagery training also included verbal presentations by the trainer of affective and action-oriented imagery including both standard and personal scripts (e.g., personally-developed scenes). "Good imagers" demonstrated physiological activity that varied with the content of the imagery instructions (i.e., action, fear, anger), suggesting that training enhanced fundamental,

emotion-specific physiological reactivity. Alternatively, "poor imagers" evidenced an overall lack of response to the standard affective scripts, but demonstrated heightened reactivity to personally-relevant emotional images, following active imagery response training.

To this end, it is important for the present study to assess whether participants are self-reported "good" or "poor" imagers. To assess imagery ability, a brief 16-item self-report measure, the Vividness of Visual Imagery Questionnaire (VVIQ; Marks, 1973; See Appendix B) was given to each participant following psychophysiological assessment. Participants who are determined to be "good imagers" may evidence heightened EMG reactivity in response to exposure to light- and season-relevant stimuli as compared to self-reported "poor imagers." Addressing of the concept of "imagery ability" is important, given that our instructions to participants explicitly ask them to imagine themselves as part of the visual stimuli.

***EMG and Cognitive Theories of Depression***

Teasdale and Bancroft (1977) used five single-subject EMG experiments to obtain evidence for established cognitive models of depression. They measured depressed mood and EMG corrugator activity in a sample of outpatient psychiatric patients exhibiting mild to moderate depressive symptoms (although not necessarily clinically depressed) while they were thinking self-selected happy and unhappy thoughts. Each participant reported greater increases in depressed mood when told to think unhappy thoughts (e.g., family member who had died, the impossibility of reaching a desired goal), as opposed to thinking happy thoughts (e.g., a loved grandson, a favorite piece of music, a pleasant social evening with friends). The average mood rating was less depressed after thinking

happy thoughts, when compared to baseline. In contrast to Greden et al. (1986), results of the corrugator activity measurement indicated that every participant demonstrated higher mean corrugator patterning after the unhappy condition versus the happy condition. Consistent with prevailing cognitive theories of depression, these results suggest that self-rated depressive symptoms are consistent with thought content (e.g., unhappy thoughts reportedly made the participants "feel" more depressed). Additionally, corrugator patterning may represent an objective psychophysiological indicator for determining an individual's emotional state.

In a similar study, Jäncke (1993) measured facial EMG reactions in a sample of nondepressed introverts and extroverts to visual stimuli with positive (e.g., women's faces, naked women), negative (e.g., war scenes, facial expressions of pain/injury) and neutral (e.g., houses, trees) valence. Results showed that both personality groups had a corresponding increase in corrugator muscle activity when presented with pictures having a negative valence and an increase in zygomatic activity when pictures with positive valence were presented. Both of these studies demonstrate that thinking about negative or unhappy affective imagery results in increased corrugator activity, whereas, thinking about positive or pleasant affective imagery results in increased zygomatic muscular activity, regardless of whether participants are depressed (Schwartz et al., 1976a) or nondepressed (Jäncke, 1993; Schwartz, Brown, & Ahern, 1980).

Another previous study investigated whether exposure to black and white photographs with men producing happy or angry facial expressions chosen from "*Pictures of Facial Affect*" (Ekman & Friesen, 1976) influences one's own mood state, and whether people engage in superficial mimicking of facial expressions they are shown

(Dimberg, 1982). This question has relevance in depression research, given that facial stimuli may represent a form of social interaction, assumed to be operating sub-optimally in individuals who are depressed. Individuals who viewed pictures with angry expressions demonstrated increased corrugator muscle activity, whereas, those exposed to happy facial expressions tended to exhibit increased zygomatic facial patterning, similar to the results of Schwartz and colleagues (Schwartz et al., 1976a; 1976b; 1980). Although participants appeared to be generating facial muscle movements that were reflective of the stimuli being viewed (e.g., increased corrugator following exposure to an angry expression), one question left unanswered by the Dimberg (1982) study is whether participants demonstrated an actual resultant change in mood state upon exposure to affect-specific stimuli. In other words, did the participants actually experience the emotion reflected in the facial expression stimuli, or were they simply "mirroring" in their own facial expressions the images they viewed.

In an attempt to answer this question, Sirota and Schwartz (1982) examined left and right corrugator and zygomatic muscle activity, as well as participants' mood ratings, after nondepressed female participants read a standard list of negative, self-referent descriptions to induce transient dysphoric mood (Velton, 1968). They were then instructed to imagine scenes with depressed, elated, and neutral content (i.e., a personal scenario that would make them happy or sad when visualized). Results indicated that facial EMG measurement is a reliable and sensitive psychophysiological indicator of experienced mood and emotion. Specifically, corrugator activity was associated with increased depression and zygomatic activity was associated with increased elation.

Similarly, Ritz, Dahme, and Claussen (1999) reported that asthmatics and healthy controls experienced increased corrugator muscle activity when presented with 20 depressing color pictures and 20 negative self-referent statements that were counterbalanced and presented as slides (Velten, 1968). Slides were selected from the International Affective Picture System (IAPS; Lang et al., 1999) and included nine stimuli in each category (i.e., happy and depressing) for each mood induction method (i.e., pictures and statements) as well as two neutral stimuli. Again, these results support the cognitive model's assumption that thought content affects mood.

***EMG and Clinical Improvement***

Changes in EMG responses also have been linked to the course of improvement with depression treatment. Schwartz, Fair, Mandel, Salt, Mieske, & Klerman (1978) evaluated resting corrugator EMG as a measure of decreased depressive symptomatology occurring with administration of tricyclics and/or placebo medications, and examined the relationship between initial resting corrugator activity as a predictor of clinical depression improvement. Results showed that EMG recording of heightened resting corrugator muscle tension in a depressed sample was associated with significant improvement in depressive symptoms 2-weeks later. This supports the notion that the human face, especially patterning of the corrugator supercilii muscle, is a reliable indicator of emotional state. A second study by Carney et al. (1981) found that depressed inpatients with increased baseline corrugator EMG levels experienced a greater reduction in depressive symptoms after 2 weeks of hospitalization and antidepressant medications, further suggesting the ability of corrugator activity to predict clinical improvement. In

addition, higher resting EMG in the zygomaticus major muscle group also was found to be a reliable, positive predictor of changes in depression.

Consistent with Schwartz et al. (1978) and Carney et al. (1981), Greden, Price, Genero, Feinberg, and Levine (1984) found that higher baseline levels of facial EMG activity of both the corrugator and zygomatic muscle regions during three imagery states (e.g., happy, sad, and typical day) predicted outcomes with endogenous depressed individuals who were treated with antidepressants. However, despite the corroborating results, Greden et al. (1984) stated that both Carney et al. (1981) and Schwartz et al. (1978) studies may be confounded by Wilder's (1957) Law of Initial Values (LIV) which states that participants measured at one point in time with higher absolute values will, at a second point in time, show greater decreases. Thus, the degree of response is related to the prestimulus level (e.g., a relative decrease of 10 units from 150 to 140 would be less significant than a decrease of 10 units from 80 to 70). In general, these studies suggest that hyperarousal of the corrugator and/or zygomaticus regions may be a marker for treatment-responsiveness.

***EMG and Gender Differences***

Few studies have focused on gender differences in EMG response. Schwartz et al. (1980) and Schwartz, Ahern, and Brown (1979) found significant sex differences in elicitation of facial EMG patterns. Females demonstrated marginally higher corrugator EMG activity at rest than males. Higher facial EMG magnitude was produced, as compared to resting states, for females when imagining self-generated scenes of high happiness (e.g., inheriting a million dollars), high sadness (e.g., mother's death), high angry (e.g., hit and run accident), and high fear (e.g., car brakes fail). In contrast, men

displayed decreased affective activity in both corrugator and zygomatic muscle groups in all conditions (i.e., happy, sad, angry, and fearful). Females also generally self-reported heightened emotional experiences to all types of affect-laden stimuli. Facial EMG patterning and emotional reactions (i.e., greater corrugator region activity with "sad" ratings and increased zygomatic activity in "happy" ratings) were more closely associated in females. In addition, in conditions where there was voluntary generation of facial expression, females generated greater EMG changes post-imagery in specific regions for specific feelings. These results suggest that females may perceive emotion in others and communicate personal feelings more accurately than their male counterparts through both overt and covert facial expression patterns (Schwartz et al., 1980).

In another study (Dimberg & Lundquist, 1990), gender differences were found such that females demonstrated increased zygomatic activity upon exposure to happy facial expressions and increased corrugator activity to angry facial expressions as compared to male participants. In this design, each participant was exposed to two different males and two different females exhibiting one happy and one angry face each. Results suggest that when exposed to angry or happy facial expressions of males and females, facial EMG patterning is a solid discriminator between various affective mood states. This result is consistent with previous empirical evidence that females may be more facially-expressive than their male counterparts to affect-laden stimuli (e.g., Schwartz et al., 1980).

Given the fact that prevalence rates for depression are twice as high among women than among men (U.S. Department of Health and Human Services [USDHHS], 1993b), and prevalence rates for SAD are at least twice as high for women (Rohan &

Sigmon, 2000; Rosen et al., 1990), gender differences in facial EMG patterning should be examined further to assess: 1) whether women with SAD and S-SAD demonstrate facial muscle movements of greater magnitude than males when experiencing differing emotional states (i.e., happy, sad, and angry); 2) degree of psychophysiological sensitivity to environmental stimuli (e.g., light-relevant, season-relevant cues) between seasonally-depressed males and females; 3) and predictive utility of resting EMG in assessing changes in depressive symptomatology when comparing males and females.

***Habituation, Individual Response Stereotopy, and Stimulus-Response Specificity***

There are several basic principles of psychophysiology that may drastically affect outcome data and results if not carefully considered and planned for when conducting psychophysiological studies. Habituation, individual response stereotopy, and stimulus-response specificity can produce psychophysiological reactions that confound psychophysiologic changes of interest elicited by the intentionally-manipulated independent variables. These constructs are reviewed below.

One basic tenant common in psychophysiological research is that of habituation; decreased responding after repeated exposure to a specific stimulus, as opposed to the expected increase in responding with onset of some stimulus (Stern et al., 2001). The phenomenon is called habituation when the response is believed to be unconditioned (O'Donohue, 1998). One form of habituation that has the potential to affect research findings is short-term habituation, which occurs within a single assessment session. However, there is some evidence to suggest that a habituation response can be partially arrested if participants are asked to provide a subjective assessment (i.e., behavioral

response) of their transient mood state at slide offset. In addition, counterbalancing, or random presentation of stimuli, can reduce the effects of habituation (Stern et al., 2001).

Another important principle of psychophysiological responding pertinent to the present study is that of individual response stereotopy (Engel, 1960; Lacey & Lacey, 1958); the tendency of individuals to emit the greatest level of psychophysiological responding within the same system (e.g., EMG, SCR), regardless of the type of stimulus or stressor applied. For example, patients with chronic head and neck pain and cardiac patients may respond to any type of stressor with increased muscle tension in the neck and increased heart rate, respectively. Consequently, careful consideration should be given to the number of participants in each group.

A final influential principle in psychophysiological responding is that of stimulus-response specificity (Lacey, 1967). Stimulus-response specificity is demonstrated in certain stimulus contexts (e.g., locking your keys in the car) and elicits specific response patterns (e.g., decreased respiration and increased skin conductance), as opposed to increases or decreases within one dimension of the activation continuum (i.e., cortical, autonomic, and behavioral) among the majority of participants (Lacey, 1967). One special case of stimulus-response specificity is termed directional fractionation; a response pattern of arousal that is dissimilar across the three activation continuums (Lacey, 1967). In other words, one form of physiologic arousal may not coincide with other measures of arousal. For example, upon hearing a gunshot, an individual may experience increased cortical arousal (e.g., electroencephalogram; EEG), concomitant increases and decreases in autonomic arousal (e.g., heightened muscle tension and skin conductance and diminution of heart rate and respirations), and a corresponding reduction

in behavioral activation (e.g., “freezing” to determine where the gunshot came from; Stern et al., 2001).

Although the concepts of individual response stereotopy and stimulus-response specificity may influence all psychophysiologic investigations to some degree, minimal impact can be ensured by choosing an appropriate number of participants (to ensure that idiosyncratic responses are true outliers) and by giving thoughtful consideration when choosing the specific physiological response parameters to be assessed (e.g., SCR, SCR magnitude, and EMG; Stern et al., 2001). The potential impact of habituation, response stereotopy, and stimulus-response specificity on the current study is discussed later in the Limitations section.

## A Role for Surface Facial EMG in SAD

At present, surface facial EMG responses to affect-laden stimuli have not been explored in SAD and S-SAD samples. In the Rohan et al. (2004) study, an S-SAD sample demonstrated increased psychophysiological arousal (i.e., SCR magnitude) to low light stimuli (presumed to be unpleasant) when measuring electrodermal response. In addition, Sigmon et al. (2002) demonstrated increased skin conductance responses and SCR magnitude in SAD, S-SAD, and SAD-HX groups when exposed to videos of winter scenes (assumed to be more unpleasant than summer scenes), when compared to nonseasonal depression and control groups.

As a follow-up to these preliminary studies, there is a need to isolate light- and season-relevant stimuli to explicate the specific stimuli content that is eliciting psychophysiological and emotional response in SAD. This type of investigation will allow determination of the possible types of conditioned stimuli in SAD. Individuals

with SAD may have a higher sensitivity to winter- and/or light-related stimuli or some combination of the two (i.e., low light/winter stimuli). Over time, these previously neutral environmental cues (e.g., low light/winter season) have been repeatedly paired with the onset of depressive symptomatology including anergia, dysphoric mood, and reduced behavioral activation. Thus, learned associations may result and generalize to other harmless cues (e.g., a picture of a dreary sky). Testing this hypothesis using surface facial EMG may represent a more sensitive psychophysiological modality than skin conductance to measure emotion-specific responses in individuals with SAD. The integration of this sensitive psychophysiological measure of affect in response to stimuli that are emotionally charged (e.g., happy and sad) combined with a potentially potent marker for changes in depression (e.g., increased baseline corrugator activity) in the context of existing cognitive-behavioral and psychological models, could improve understanding of etiological and maintenance mechanisms of SAD.

Facial EMG measurements in SAD and S-SAD samples should be measured using a paradigm similar to the Rohan et al. (2004) and Sigmon et al. (2002) method. Specifically, EMG should be assessed in response to light- and season-relevant visual stimuli. If the increased EMG response found in nonseasonally-depressed individuals upon exposure to unpleasant, unhappy, or negative stimuli generalizes to seasonal depression, SAD samples should demonstrate similar increases in EMG corrugator arousal to low light and winter season stimuli. This methodology also may reveal increased EMG zygomatic activity to bright light and summer season stimuli among individuals with SAD. This is an important contribution of EMG studies over skin conductance, which only assesses negative, aversive, or unpleasant reactions.

***Study Justification***

The purpose of this study is to investigate whether individuals with SAD demonstrate different patterns of surface facial electromyography (EMG) reactions when exposed to visual stimuli that vary in light intensity and seasonal cues. This study will examine specific facial muscle group activity, namely the corrugator supercilii (i.e., brow-pursing, correlated with the frown response) and the zygomaticus major (i.e., muscles involved in the smile response), as well as significant SCRs and SCR magnitude in reaction to light- and season-relevant stimuli. Using both skin conductance and EMG as measures allows separation of general sympathetic arousal (as assessed by skin conductance) from emotion-specific reactivity (as measured by facial EMG) to the stimuli. Subjective self-report ratings of affective state in response to environmental stimuli will also be assessed in the present study. This will allow examination of whether participants' self-reported ratings of mood state are consistent with their pattern of facial muscle activity.

Comparisons will be made between two groups (i.e., SAD and controls) based on the spectrum of disease model suggesting that the expression of depression in a seasonal or nonseasonal pattern is dependent upon whether an individual has "primary loading" on the depression factor, the seasonality factor, or an intermediate loading on both factors (Lam et al., 2001). Thus, the current study will compare a SAD group that presumably has a primary vulnerability to seasonality, and a secondary vulnerability to depression, and a never-depressed control group that has neither a vulnerability to seasonality nor to depression.

Given the previously reviewed results (Rohan et al., 2003, 2004; Sigmon et al., 2002), there may be some type of vulnerability factor for SAD onset and maintenance involving classically conditioned reactions to, or possible negative cognitive processing (including activation of negative core schemas; Beck, 1967; 1976) in response to low light and winter season-relevant cues. This cognitive bias or learning history may result in increased psychophysiological response upon exposure to light- and season-related stimuli. Examination of surface facial EMG response in SAD may help explicate whether individuals with SAD show increased psychophysiological reactivity to light-relevant stimuli, season-relevant stimuli, or an interaction of the two. Perhaps the combination of low light and winter stimuli will result in the most intense corrugator EMG response and depressed mood, whereas the combination of bright light and summer stimuli will result in the most exaggerated zygomatic activity and least dysphoria in SAD.

## HYPOTHESES

In testing the following hypotheses, analyses will be conducted using individuals diagnosed with Major Depressive Disorder, Recurrent with Seasonal Pattern Specifier who also meet criteria for a current SAD episode (SAD) and nonseasonal, nondepressed controls.

### *Hypothesis 1: Corrugator Supercilii Activity in Response to Low Light and Winter Visual Stimuli*

SAD participants will demonstrate increased corrugator muscle reactivity (i.e., elicitation of a brow-pursing, correlated with a frown response) in response to low light/winter season stimuli relative to control participants. A significant interaction between group status and stimulus characteristics is hypothesized, such that the greatest

corrugator reactivity will be observed in the SAD participants in response to the low light/winter condition as compared to the bright light/summer condition. In addition, a main effect for group (SAD and control) is hypothesized as well as main effects for stimulus characteristics (brightness: low light versus bright light, and season: winter versus summer and fall).

***Hypothesis 2: Zygomaticus Major Activity in Response to Bright Light and Summer Visual Stimuli***

SAD participants will demonstrate increased zygomaticus muscle reactivity (i.e., elicitation of a smile) in response to bright light/summer season stimuli relative to control participants. A significant interaction between group status and stimulus characteristics is hypothesized, such that the greatest zygomatic reactivity will be observed in the SAD participants in response to the bright light/summer condition as compared to the low light/winter condition. Furthermore, a main effect for group (SAD and control) is hypothesized as well as main effects for stimulus characteristics (brightness: bright light versus low light, and season: summer versus fall and winter).

***Rationale for Hypotheses 1 and 2***

These hypotheses are based on the central hypothesis that through learned associations, cues associated with low light availability and winter become more salient and unpleasant and cues indicative of bright light availability and summer become more salient and pleasant to individuals with SAD relative to control participants (Rohan et al., 2004; Sigmon et al, 2002).

***Hypothesis 3: Significant Skin Conductance Response and Skin Conductance Response Magnitude to Low Light and Winter Visual Stimuli***

SAD participants will demonstrate more frequent significant skin conductance responses (SCR) and greater SCR magnitude in response to low light/winter season stimuli relative to control participants. A significant interaction between group status and stimulus characteristics is hypothesized, such that more significant SCRs and the greatest SCR magnitude will be observed in the SAD participants in response to the low light/winter condition and fewer significant SCRs and the lowest SCR magnitude in response to the bright light/summer condition. In addition, a main effect for group (SAD and control) is hypothesized as well as main effects for stimulus characteristics (brightness: low light versus bright light, and season: winter versus summer and fall).

***Rationale for Hypothesis 3***

Rohan et al. (2004) found that women with S-SAD demonstrated higher skin conductance response (SCR) magnitude than controls in response to low light stimuli during winter and greater SCR magnitude in response to low light scenes relative to bright light or ambiguous light scenes. In another study, participants with any significant history or current seasonal depression (i.e., SAD, S-SAD, and SAD-HX) exhibited more significant skin conductance responses (SCRs) and increased SCR magnitude to winter video scenes when compared with MDD and control groups (Sigmon et al., 2002).

***Hypothesis 4: Depression-Dejection Subscale Scores on the Profile of Mood States and Lower Perceived Pleasantness Ratings for Low Light and Winter Visual Stimuli***

SAD participants will experience increased transient depressed mood state, as assessed by self-report on the POMS Depression-Dejection subscale, in response to low

light/winter season stimuli relative to control participants. A significant interaction between group status and stimulus characteristics is hypothesized, such that SAD participants will report the greatest increase in depressed mood state (from baseline) on the POMS in response to the low light/winter stimuli and the greatest reduction in depressed mood subsequent to presentation of the bright light/summer stimuli. An interaction is also hypothesized such that SAD participants will report lower perceived levels of pleasantness in response to the low light/winter condition as compared to the control group, and will describe the low light/winter stimuli as less pleasant than the bright light/summer stimuli. Finally, a main effect for group (SAD and control) is hypothesized as well as main effects for stimulus characteristics (brightness: low light versus bright light, and season: winter versus summer and fall).

***Rationale for Hypothesis 4***

Previous studies suggest a heightened emotional reactivity to light-related stimuli in SAD. Women with a history of SAD (Rohan et al., 2003) and women with S-SAD (Rohan et al., 2004) reported an exacerbation of depressed mood in response to low light stimuli as compared to bright- and ambiguous-light stimuli. In these studies, SAD and S-SAD women reported a greater improvement in baseline depressed mood after exposure to bright light slides relative to controls. Sigmon et al. (2002) demonstrated that participants in SAD and MDD groups reported more depressed mood in reaction to winter scenes than to summer scenes when compared to individuals in the SAD-HX and control groups.

## DATA ANALYTIC STRATEGY

All hypotheses (1 through 4) will be tested using 2 (participant group; SAD, nondepressed controls) x 2 (light intensity; low light, bright light) x 3 (season; summer, fall, winter) ANOVAs. The ANOVA dependent variables will be: corrugator and zygomatic EMG mean and peak responses, SCR and SCR magnitude, POMS Depression-Dejection subscale ratings, and ratings of perceived pleasantness. For this pattern of findings, a significant Group X Light Intensity X Season interaction is predicted. If found, the 3-way interaction will be followed up with tests of two-way interaction effects examining the Group X Light interaction within each level of season. If a 2-way interaction is found, simple main effect analyses will be performed followed by post-hoc analyses with a Bonferroni correction.

For EMG corrugator mean and peak response, SCR response, SCR magnitude, POMS Depression-Dejection subscale scores, and ratings of perceived pleasantness, a significant Group X Light interaction within winter season is predicted. If an interaction is found, it will be followed up with simple main effect analyses to examine the main effect of Group within winter season stimuli for low light slides. Here, a significant Group main effect is predicted with SAD > Controls on the low light, winter slides. In addition, pre-planned post-hoc linear contrasts will be used to compare low light/winter slides to bright light/summer slides within the SAD group. A significant difference is expected with low light/winter > bright light/summer.

For EMG zygomatic mean and peak response, a significant Group X Light interaction within summer season is expected. If an interaction is found, simple main effect analyses will be conducted to examine the main effect of Group within summer

season stimuli for the bright light slides. Significant group differences are expected with SAD > Controls on zygomatic responses to bright light, summer stimuli. Pre-planned post-hoc linear contrasts will compare EMG zygomatic reactions to low light/winter versus bright light/summer stimuli within the SAD group. Here, SAD participants are expected to demonstrate significantly greater zygomatic activity to bright light/summer than to low light/winter slides.

For each analysis, effect size (i.e., partial $\eta^2$) will be reported. In addition, Cohen's (1988) definition of small (.10), medium (.25), and large (.40) effect size cut-offs will be applied. Wilks' Lambda will be reported for all analyses, as it is the standard statistic used in clinical psychology empirical research.

## METHOD

### *Participants*

***Inclusion Criteria.*** Inclusion criteria for the SAD group include a diagnosis of Major Depression, Recurrent, with a Seasonal Pattern on the SCID.

***Exclusion Criteria.*** Exclusion criteria for the study are: 1) any co-occurring Axis I Disorder; 2) current participation in, or plans to initiate light therapy, psychotherapy, or other psychiatric treatment (e.g., psychotropic medications); and 3) any extended period of absence throughout the upcoming winter season outside of the study area. If at any time while enrolled in the treatment study a participant indicates that they have plans to initiate antidepressant medication therapy, the participant will be allowed to continue in the treatment study and the data will be analyzed both with and without their data. However, based on our experience in this type of treatment study, pharmacotherapy

initiated after commencement of treatment has not been an issue with previous participants.

***Recruitment.*** Adult male and female participants will be recruited through media advertisements in the greater Washington D.C. metro area. Upon response to advertisements, participants will undergo a brief phone screening to ascertain whether they may qualify for the study. This involves brief assessment of DSM-IV (APA, 1994) criteria for MDD and seasonality. Participants must meet diagnostic criteria for SAD because they are being recruited for a treatment study which will commence following the pre-treatment assessment (e.g., facial EMG and SCR assessment). Those respondents who do not meet phone screen criteria for possible inclusion in the study will be sent a referral list of local mental health providers. Individuals who may be eligible to participate based on the phone screen will be invited to our laboratory to review the informed consent document and undergo a thorough diagnostic interview, the Structured Clinical Interview for DSM-IV Axis I Disorders – Clinician Version (SCID-CV; First, Spitzer, Gibbon, & Williams, 1995). Based on the SCID, appropriate individuals will be identified for the SAD group (i.e., primary diagnosis of Major Depressive Disorder, Recurrent, with Seasonal Pattern Specifier) or the nondepressed, non-SAD control group (i.e., no diagnosis). Individuals who do not qualify based on the SCID will also receive the referral list of local mental health providers. In order to ensure 30 "completers" in the SAD group, we will need to recruit approximately 35 participants based on our experience with participant drop-out and withdrawal. We plan to include 30 SAD and 30 control participants for this study.

*SAD Participants.* Over the last two winters, each SAD participant must have experienced a Major Depressive Episode that commenced in the fall/winter and remitted the following spring, which was not better accounted for by seasonally varying psychosocial stressors (e.g., holiday season; Rosenthal et al., 1984b). In addition, SAD participants must meet the Structured Interview Guide for the Hamilton Rating Scale for Depression – Seasonal Affective Disorder Version (SIGH-SAD; Williams, Link, Rosenthal, Amira, & Terman, 1992) criteria for a current SAD episode. Terman et al. (1989) defined criteria for SAD episode onset and relapse as: total SIGH-SAD score $\geq$ 20 + the 21-item Structured Interview Guide for the Hamilton Rating Scale for Depression (HAM-D) score $\geq$ 10 + 8-item atypical score $\geq$ 5.

*Nonseasonal, Nondepressed Control Participants.* Control participants must be free of current axis I psychopathology and have no history of past Major Depressive Episodes on the SCID. In addition, controls must score $\leq$ 10 on the Beck Depression Inventory – Second Edition (BDI-II; Beck, Steer, & Brown, 1996) and score in the normal range on the Modified Seasonal Pattern Assessment Questionnaire (M-SPAQ; Lam, Goldner, & Grewel, 1996): a) global seasonality score (GSS) of 8 or 9, but no problems across the seasons; or b) GSS $\leq$ 7.

### *Measures*

***Assessment of Menstrual Cycle Phase (AMCP).*** The AMCP is a brief, 4-item descriptive questionnaire designed for this study to ascertain the current phase of the menstrual cycle for all female participants (See Appendix A). The measure includes an item assessing whether female participants have reached menopause; if not, the questionnaire inquires about the date when the last menstrual cycle began and the average

cycle length in days. Finally, the questionnaire ascertains whether female participants are using birth control and the method. This question is necessary given that certain forms of birth control (e.g., Norplant, Depo-Provera) often leave women amenorrheic for several cycles.

***Profile of Mood States (POMS).*** The Profile of Mood States Depression–Dejection Subscale (POMS; McNair et al., 1971), a subjective state measure of transient depressed mood is a 15-item subscale from the original 65-item measure (See Appendix C). In this study, participants are also given the 9-item POMS Tension–Anxiety subscale in order to mask the fact that we are interested in subjective report of depressed mood. Participants indicate the degree to which they are currently feeling each emotion (e.g., sad, unworthy, hopeless) on a 5-point Likert scale ranging from 0 = "not at all" to 4 = "extremely." The POMS Depression-Dejection subscale has good psychometric properties, including an internal consistency of .95 in two distinct studies (Butcher, Dahlstrom, Graham, Tellegren, & Kaemmer, 1989; Nezu, Ronan, Meadows, & McClure, 2000). In addition, the POMS has been found to correlate highly with other measures of depressive symptomatology, including the BDI ($r$ = .61; Beck et al., 1996) and the MMPI-D scale ($r$ = .65; Butcher et al., 1989; Nezu et al., 2000).

***Perceived Pleasantness Rating Scale.*** The perceived pleasantness rating scale (See Appendix D) is a brief 1-item measure on which participants rated each block of slides (e.g., using a 1 = "very pleasant" to 7 = "very unpleasant" Likert scale) delineating the degree of pleasantness they perceived for each slide type (e.g., bright light/summer and low light/winter).

***Vividness of Visual Imagery Questionnaire (VVIQ).*** The VVIQ is a brief, 16-item measure of visual imagery ability (White, Sheehan, & Ashton, 1977; See Appendix B). Items include four aspects of familiar scenes (e.g., a friend or relative, the rising sun, a country scene) for vividness rating on a 5-point Likert rating scale ranging from 1 = "perfectly clear and as vivid as normal vision" to 5 = "no image at all, you only 'know' that you are thinking of something." One distinguishing feature of the VVIQ is that all items can be rated twice; once with the eyes open and once with the eyes closed. However, psychometric research studies have shown that there is no significant difference between the two scores, therefore, only the eyes open ratings will be obtained in the present study. The VVIQ is also significantly correlated ($r$ = .67) with other imagery measures (e.g., Gordon's Test of Visual Imagery Control; TVIC), which assesses the degree of difficulty associated with control or manipulation of visual imagery. Studies have also shown the VVIQ to have good split-half reliability ($r$ = .85) and a high alpha ($\alpha$ = .89) which indicate acceptable internal consistency (McKelvie, 1995; White et al., 1977). Additionally, the VVIQ demonstrates good immediate test-retest reliability ($r$ = .94), but marginally-acceptable delayed test-retest reliability ($r$ = .74), which suggests that the test should be administered in close proximity to any predicted criterion (McKelvie, 1995), as is the case in the present study.

### *Psychophysiological Task*

***Pilot Study 1 – Selection of Stimuli.*** Pilot Study 1 was conducted to select the slides for inclusion in the task. Participants were a convenience sample of male and female graduate students and professors at the Uniformed Services University of the Health Sciences at the conclusion of a research seminar ($N$ = 22). Twenty-four

environmental slides of digital photographs were projected one at a time on a screen. The 24 slides consisted of 12 pairs with each pair including the same stimulus scene shown under sunny conditions and under dark, dreary weather conditions. The photographs were taken at local parks. For the purposes of the pilot, we used only summer season photographs. Summer slides depicted environmental stimuli (e.g., bushes, trees) that were covered in green foliage or in bloom.

Participants were asked to give each slide two ratings. The first was a rating of emotion elicited by the slide, given on a 7-point Likert scale where 1 = "very sad" and 7 = "very happy." The second was a rating of light intensity, where participants assessed the intensity of light present in the photograph. Light ratings were also made using a 7-point Likert scale with 1 = "dark/dreary" and 7 = "bright/sunny."

Using the pilot data, Pearson correlations were computed to determine whether light ratings were correlated with emotion ratings within each type of photograph (i.e., sunny and dark). We expected that both bright/sunny stimuli and dark/dreary stimuli would generally demonstrate positive correlations between the light and emotion ratings. When examining Likert pleasantness ratings in reaction to slides of varying light intensities, Rohan et al. (2004) found that regardless of the time of year, all participants (i.e., S-SAD and controls) rated bright light slides as more pleasant than either low light or ambiguous light slides and rated ambiguous slides as more pleasant than low light slides. Therefore, the correlations in this study were based on the assumption that, in the general population, bright light stimuli elicit more positive emotional reactions and low light stimuli elicit more negative emotions (Rohan et al., 2004), resulting in positive correlations between light intensity and emotion ratings for both light intensities.

This procedure also allowed identification of any scenes that were negative or positive, regardless of light intensity, in the case of a negative correlation. Following determination of which slides showed the highest positive correlations between emotion and light ratings (i.e., the sunny slides with the highest light ratings and the highest emotion ratings and the low light slides with the lowest light ratings and the lowest emotion ratings), the five pairs of slides from the original 12 pairs with the highest light/emotion correlations were chosen as appropriate stimulus "pairs." These scenes represent the "best" stimuli for discerning "light" and "emotionality" ratings from a non-clinical convenience sample of pilot participants. For the selected light slides, correlations between light and emotion ratings ranged from $r = .60$, $p < .01$ to .77, $p < .01$, whereas correlations for the selected low light slides ranged from $r = .11$, *ns*, to .67, $p < .01$. Therefore, results from pilot study 1 demonstrate that the five scenes chosen for the current study have the strongest positive relationship between light intensity ratings and mood state.

Following selection of the five pairs of slides, additional digital photographs were taken to capture each scene in the other seasons (i.e., fall and winter) and under both light intensity conditions (i.e., on a bright/sunny day and on a dark/dreary day). In the task, each of the five pairs of slides is represented in all six conditions (i.e., bright light/summer, low light/summer, bright light/fall, low light/fall, bright light/winter, and low light/winter). Fall photographs presented environmental stimuli that were clearly changing colors (e.g., red, orange, and yellow leaves), illustrating the onset of the autumn season. Finally, winter photographs were of the same environmental stimuli without any foliage (e.g., bare trees, bushes), clearly representing the stark winter landscape.

***Pilot Study 2 – Validation of Stimuli***. In order to validate the selected stimuli, a second pilot study was conducted to examine transient mood state reactions to the stimuli, as assessed by the POMS (McNair et al., 1971), among individuals with SAD. In addition, SAD participants rated each block of slides according to their degree of pleasantness on a 7-point Likert scale. The light- and season-relevant slides were piloted on a sample of participants recruited for a treatment study who met DSM-IV (APA, 1994) diagnostic criteria for SAD ($N$ = 15). One participant's scores were excluded from the analyses because they were a significant outlier (i.e., scores were greater than two standard deviations from the mean). The 30 environmental slides were presented on a computer screen in blocks according to light intensity and season (i.e., five bright light/summer, five low light/summer, five bright light/fall, five low light/fall, five bright light/winter, five low light/winter). The order of the blocks of slides was randomized across participants (See Appendix E). Prior to reviewing the slides, participants were asked to complete an abbreviated POMS, including the Depression-Dejection and Tension-Anxiety subscales.

Each environmental slide was presented for a period of 10 seconds with a 10-second slide-offset period. Participants were instructed as follows: *After a 5-minute resting period, I will ask you to complete this questionnaire to rate your mood. Then you will view several photographs on the computer screen of various outdoor scenes. Each photograph will be shown for a few seconds, followed by a blank screen for a few seconds. I would like for you to relax and imagine what it would feel like if you were actually in the picture. Imagine what you would be feeling and thinking if you were really there. After you look at five similar pictures, the computer screen will pause, and I*

*will ask you to rate that group of pictures considering how pleasant or unpleasant they were for you on this 7-point rating scale and to rate your mood again. I will then restart the computer and the process will be repeated five more times, with five different groups of similar pictures. Do you have any questions or concerns before we start?*

In Pilot Study 2, POMS Depression-Dejection subscale change scores demonstrated fluctuating mood states in response to light- and season-relevant environmental stimuli in the predicted direction. However, because there was a large degree of individual variance on POMS change scores within the SAD group, median statistics were used to provide a better estimate of the distribution of the data (Keppel, 1991). Participants generally reported improved mood from baseline on the POMS immediately after viewing blocks of bright light/summer slides (median = 5.0) with a mean of 5.87 (95% CI: 2.30 - 9.44). In contrast, after viewing low light/winter slides (median = -3.0) with a mean of -2.40 (95% CI: -6.31 - 1.51), participants generally demonstrated exacerbated depressed mood (See Table 2). Given that the bright light/summer and low light/winter slides were not normally distributed, Wilcoxon signed-ranks test were performed and the slides were statistically different at $p < .01$.

When examining self-reported pleasantness ratings of the environmental stimuli, the between-subjects variance was significantly lower. Therefore, means and standard deviations are reported. SAD participants rated all blocks of bright light slides (bright light/summer $M = 5.17$, $SD = 1.47$; bright light/fall $M = 4.67$, $SD = .78$; and bright light/winter $M = 4.25$, $SD = 1.36$) as more pleasant than blocks of low light slides (low light/summer $M = 3.58$, $SD = 1.56$; low light/fall $M = 3.25$, $SD = .87$; and low light/winter $M = 2.42$, $SD = 1.08$), regardless of season (See Table 3). A *t*-test was

performed on the pleasantness ratings of the bright light/summer slides and the low light/winter slides resulting in a significant difference at $p < .001$. Consequently, it appears as if various light intensity and seasonal slides in the present study represent valid stimuli as per Tables 1 and 2 for measuring emotion-specific reactivity in a SAD sample.

***Facial Muscle Electrode Placement.*** In accordance with existing EMG literature (Tassinary, Cacioppo, Geen, & Vanman, 1987; Tassinary et al., 1989; Fridlund & Cacioppo, 1986), surface facial EMG activity was recorded using the zygomaticus major, the muscle primarily responsible for production of a smile, and the corrugator supercilii, the main muscle involved in lowering the brow while frowning, on the left side of each participant's face for standardization with SCR measurement. Electrode placement was arranged in pairs, and for the corrugator muscle was situated at a 60° angle to the facial midline, approximately 1 cm above the eyebrow and 2 cm to the right of the nasal midline. Both mean and peak corrugator responses were recorded on channel 2. Two electrode pairs were placed to record left zygomatic muscle activity, incorporating both the upper and lower zygomaticus channels. The first electrode pair was placed about 2.5 cm from the base of an imaginary line drawn between the corner of the resting lip (i.e., cheilion) and the middle portion of the front of the ear (i.e., ipsalateral condylion). The second electrode pair was placed posterior and lateral to the first along the imaginary line extending from the cheilion to the condylion (See Appendix F). Within the zygomaticus muscle, two EMG channels were used to simultaneously record activity (i.e., channel 3 – lower zygomatic; channel 4 – upper zygomatic). Both mean and peak responses were assessed on both the lower and upper zygomatic muscles.

***Skin Preparation.*** Prior to attachment of electrodes, each participant's face was washed with a wet facial cleansing cloth and alcohol in the areas where the electrodes were placed, and then lightly abraded with Redux paste swirled on the skin for 20 seconds with a cotton swab, in order to reduce skin resistance. Optimal impedance levels for the pair of electrodes can vary from 5 kΩ to 10 kΩ, either of which represents a reliable connection (Cutmore & James, 1999; Dow, 1991). Impedance levels greater than 10 kΩ would reduce the strength of the signal sent to the amplifier, and could cause interference (i.e., noise). An impedance check was performed for each participant with the active electrode input terminal for each channel providing a 30 Hz 0.5 μV square wave signal. A 50 mV Full Scale Deflection (FSD) was expected. Impedance levels of < 10 kΩ were considered acceptable (i.e., green indicator light for < 5 kΩ and yellow indicator light for < 10 kΩ), whereas, any impedance measurements indicated at a level > 10 kΩ (i.e., orange indicator light) required the experimenter to remove and reapply the electrodes after further skin abrasion was completed.

The paste was left on the skin for 1-2 minutes and then removed with a cloth. Double-sided adhesive collars were attached to miniature Ag-AgCl electrodes filled with electrolyte gel, with surfaces 0.40 cm in diameter, and placed directly on the facial skin. Surface facial EMG patterning was recorded in response to light- and season-relevant environmental stimuli using PSYLAB software (Dow, 1991) operated on Contact Precision Instruments psychophysiological equipment (London, United Kingdom). Data was digitized using PSYLAB Stand Alone Monitor (SAM) providing a 12-bit high speed analog-to-digital converter (ADC) on a maximum of 16 channels at < 1.5 kHz installed on a Gateway computer (Intel 800MHz Pentium III processor). Prior to commencement

of psychophysiological recoding, the EMG signal was calibrated via an internal calibration signal, thereby allowing between-subjects quantification of EMG responses. The Contact Precision Instrument's active electrode input terminal of all channels commenced a low impedance 1 Hz 100 μV square wave pulse, while the reference terminal was changed to "isolated ground" potential. This calibration step served to examine the entire amplifier circuit with a simulated electrode signal of 100 μV which was assessed at the ATD converter.

The raw EMG signal was filtered and amplified via the BIO2 wide bandwidth Bio-amplifier. The gain, or FSD, was 100 μV, inputting an AC signal into the electrode and measuring the resulting voltage with an ADC range of 6 V (i.e., nominal output voltage +/- 3V). This FSD setting was important so that the output did not exceed the limits of the equipment, which would result in "clipping" of the data, and inaccurate recording of the biological signal (Cutmore & James, 1999). Amplification of surface facial EMG necessarily dictates the following: high gain, high input impedance, and frequency response from 1 to 1,000 Hz (Stern et al., 2001). High gain was necessary because EMG signals are both low voltage and low current (Cacioppo, Tassinary, & Berntson, 2000). The primary energy in bipolar surface facial EMG recording is found between 10 and 200 Hz (Cacioppo et al., 2000). In the present study, the EMG passband for analysis was from 80 to 250 Hz (a passband from 10 Hz to 500 Hz is sufficient for the majority of psychophysiological recording circumstances; Cacioppo et al., 2000). Because of this passband, the filter settings included a low pass filter (LPF) setting of 500 Hz to include the full spectrum of EMG energy, and a high pass filter (HPF) of 30 Hz, ideal for EMG measurement (Dow, 1991).

The most common noise interference is found at 60 Hz and can be emitted by electrical equipment, fans, computers, and fluorescent lights (Cutmore & James, 1999). In the present study, protective shielded ribbon cable, a high common mode rejection ratio (i.e., 110 dB), and a higher input impedance of the BIOAMP aided in reducing interference for obtaining a better signal. A selectable twin T active notch filter was turned on for every participant providing 50 dB octave hum reduction at 50 or 60 Hz. Although the notch filter significantly reduced interference, it also caused reductions of the EMG energy. In addition, the system incorporated an "un-blocking" facility that allowed for re-centering of the EMG trace immediately prior to a measurement trial, or at any time when large movement artifacts resulted in the signal extending beyond the specified range. This function temporarily engaged the 100 Hz HPF thereby removing blocked potentials from the data within a maximum of 0.05 seconds (T = 1.6 mS).

The sampling rate for EMG potentials in this study was 1,000 Hz (i.e., twice the maximum frequency target component or LPF) as per the Nyquist sampling theorem (Cutmore & James, 1999). This allowed sampling at a rate fast enough to fully characterize the signal and noise, and allowed accurate quantification of the data such that high frequency noise was reduced to a negligible level prior to digitization of the EMG signal (Cutmore & James, 1999). Finally, the Contact Precision Instruments system incorporated an anti-aliasing filter set at 33% of the sampling frequency to eliminate large frequency "noise" components higher than the sampling rate chosen for the study which may have given a false representation of the EMG signal (Cutmore & James, 1999).

***Psychophysiological Data Reduction.*** The EMG signal was first recorded as a raw EMG signal. However, in order to perform a quantitative analysis of the data, the continuous analog signal was converted into a digital signal via the ADC. After digitization of the signal, rectification and low pass filtering, as well as smoothing (also referred to as integration), were automatically performed by the system. The rectification process is one in which positive and negative values become combined around a specific value (Stern et al., 2001). Essentially, rectification is the sum of the continuous areas above and below the EMG trace over time, representing absolute EMG amplitude at any point in time. When used with PSYLAB software, SAM was set to Rectifier/Integrator mode. In this configuration, the SAM ADC operated at a high data rate (i.e., approximately 10 kHz), with each sample rectified and added to the previous reading. This value was then divided by the number of samples taken within the specified timeframe defined for each sample sent to the data file.

Following rectification, smoothing of the data took place allowing quantification of EMG peaks or assessment of the area under the curve. In this assessment, EMG response occurred simultaneously with the presentation of stimuli (e.g., within 1 to 3 mS), and also occurred throughout the prolonged stimulus presentation (i.e., 10-second slide onset). Therefore, in the present study, analyses were conducted on mean response and peak response (i.e., amplitude) to each type of stimulus (e.g., light/fall; Stern et al., 2001). This reliable EMG potential was then directly compared to potentials obtained under alternative stimulus conditions (e.g., dark/winter).

***Skin Conductance.*** Skin conductance was assessed using Contact Precision Instruments psychophysiological equipment (London, UK) with PSYLAB software

(Dow, 1991) which provided 24-bit accuracy ADC built into the amplifier, such that skin conductance data was digitized as early as possible, thereby rendering the data immune to any further interference. Automatic evaluation of SCR in the stimulus-response paradigm allowed phasic response to be analyzed at stimulus onset, onset latency, peak latency, and amplitude. The psychophysiological system measured skin conductance directly using DC coupling with constant voltage electrode excitation (Dow, 1991). The high resolution of the system allowed SCRs below 0.01 micro-Siemen to be amplified, rectified, and smoothed via the software. In this study, skin conductance response was measured in two ways. Logarithmic transformations were calculated for baseline skin conductance level (SCL) to reduce skewing effects caused by outliers (Venables & Christie, 1980). The first skin conductance measure, significant skin conductance response (SCR), was defined as a change of at least 0.05 micro-Siemen from baseline SCL summed within type of stimulus (Venables & Christie, 1980). The second measure, SCR magnitude, was determined by adding 1 to the SCR with the largest response and taking the log (Venables & Christie, 1980).

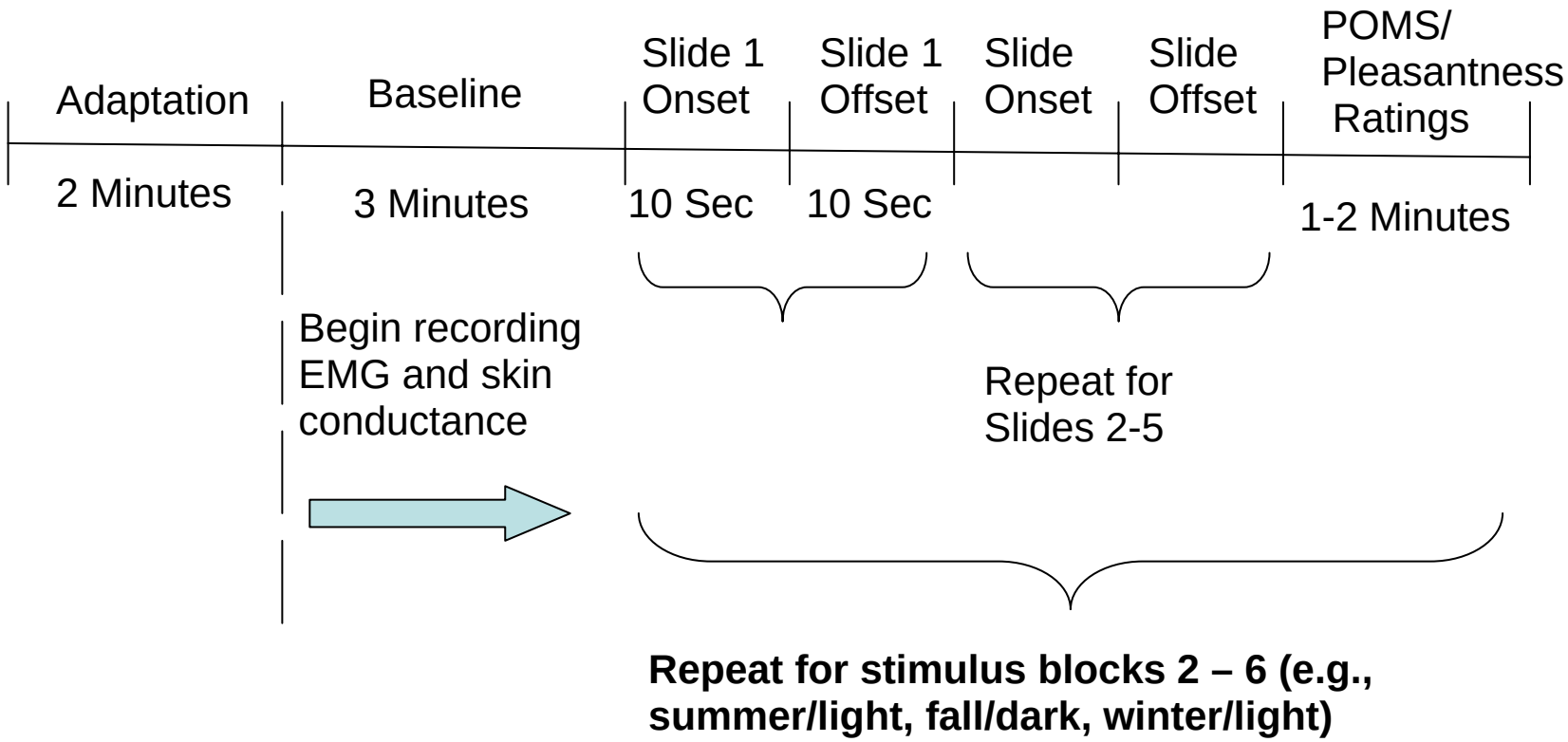
To obtain the skin conductance measurement, electrodes were placed in a bipolar arrangement involving the medial phalanx of the third (i.e., middle) and fourth (i.e., pointer) fingers on the participants' non-dominant hand. Electrodes filled with EC22 paste for skin resistance and conductance were secured in place with GRASS EWS-500 (25 mm or 1 inch) electrode collars (Warwick, RI). In addition, surgical tape was wrapped comfortably around the fingers to further secure the electrodes. Each participant's hand was placed on the arm of an easy chair with the palm facing upward to prevent artifact. Prior to SCR measurement, automatic calibration took place switching

from 0 micro-Siemen (open circuit) to 100 micro-Siemen (0.1% 10 kΩ precision resistance). Upon calibration completion, SCL readout was visible in the LCD display window. A 10 Hz filter was applied to the SCR response signal to prevent aliasing. In addition, the Pre-amplifier power box of the Precision Instruments equipment (London, United Kingdom) sample rate was set at 40 Hz (Dow, 1991).

***Recording Procedure.*** After connecting all electrodes, participants were instructed to relax and sit quietly in a comfortable chair during a 2-minute adaptation period and a 3-minute baseline recording period (See Figure 5). Following baseline, participants completed a POMS, and then were presented with the six blocks of environmental slides of differing light intensities and seasonal content (i.e., bright light/summer, low light/summer, bright light/fall, low light/fall, bright light/winter, and low light/winter) with a total of five slides in each block. The order of each stimulus-type block was counterbalanced across individuals (See Appendix E).

***Figure 5.***

**Facial EMG Task Timeline.**



Each stimulus was displayed for a period of 10-seconds followed by another 10-second post-stimulus interval in which facial EMG mean and peak response, SCR, and SCR response magnitude were recorded. Just prior to presentation of each block of slides, individuals were instructed to imagine how they might feel if they were actually present in the displayed environment using explicit directions to the participants (See Appendix F). For each of the six blocks of stimuli, skin conductance and EMG were recorded during both the 10-second interval during and following slide presentation. Respiration, EKG, and temperature were also recorded simultaneously so that participants did not focus exclusively on either area of interest (i.e., facial muscle movement, finger sweat production), thereby, potentially affecting EMG or SCR outcome data. However, the data from these psychophysiological measures were not analyzed in the present study. Slide presentation was interrupted briefly after each block of slides and psychophysiological recording was paused for approximately 1 to 2 minutes so that participants could complete a subjective measure of transient mood state (i.e., POMS Depression-Dejection subscale and Tension-Anxiety subscale) and make a subjective rating of how pleasant the previous slides were on a 7-point Likert Scale ranging from 1 = “very unpleasant” to 7 = “very pleasant” after viewing blocks of low light versus bright light environmental slides. Participants were asked to move as little as possible while completing the POMS.

***Timing of the Study Procedures***

SAD participants completed the psychophysiological task during a fall/winter month prior to their enrollment in an ongoing randomized clinical trial (Rohan, 2002). Control participants completed the psychophysiological task immediately following

recruitment and SCID diagnosis. This study was approved by the Institutional Review Board at the Uniformed Services University of the Health Sciences.

***Power Analysis***

Two prior studies examined SCR and SCR magnitude in seasonality samples. Sigmon et al. (2002) compared SAD, MDD, S-SAD, SAD-HX, and control groups on skin conductance reactions to seasonal stimuli and found significant differences at the .05 level when comparing SAD and MDD ($M$ = 3.31, $SD$ = 3.88) and when comparing SAD ($M$ = 11.69, $SD$ = 6.41) and controls ($M$ =3.60, $SD$ = 3.78) on skin conductance response. When examining SCR response magnitude, significant differences at the .05 level were found when comparing SAD and MDD ($M$ = 4.21, $SD$ = 3.06) and when comparing SAD ($M$ = 7.36, $SD$ = 1.99) and controls ($M$ = 4.72, $SD$ = 3.08). Rohan et al. (2004) compared S-SAD and control groups on skin conductance reactions to light-related stimuli and found significant differences at the .05 level when comparing S-SAD ($M$ = 2.73, $SD$ = 1.98) and controls ($M$ = 1.33, $SD$ = 1.32) on skin conductance response. When examining skin conductance response magnitude, Rohan et al. (2004) found significant differences at the .05 level when comparing S-SAD ($M$ = 2.55, $SD$ = .38) and controls ($M$ = 1.37, $SD$ = 1.04).

Power analyses were calculated using nQuery Advisor (Elashoff, 2000), and were based on a one-way analysis of variance between-groups tests (equivalent to the 2-group *t*-test), comparing SAD participants versus controls. Sample size calculations were not conducted based on the above mentioned 2 x 2 x 3 ANOVA because little is known about the inter-correlation of the repeated measures (light and season), precluding precise effect size estimation. Based on the findings described above, an effect size ($\eta^2$) of 0.70 was

postulated ($\mu_{:1} - \mu_{:2} / \Phi_{12}$) = 0.70) for the comparison between SAD versus control participants. A $\eta^2$ of 0.70 corresponds to an $\omega^2$ of .17, which has been described as a large effect size (Cohen, 1977); however, in the present study, the effect size realized was not large, given the significant variability common in psychophysiological research (Cacioppo et al., 2000). A sample size of 26 participants per group would suffice to detect a difference between the SAD and control groups with an effect size of 0.70 at a one-sided alpha level of 0.05 (directional hypothesis) and a power of 80%. The estimated power of the proposed study was larger than 80%, because the power analysis did not include the repeated measures component of the design.

When considering the POMS, change scores from baseline for bright light/summer versus low light/winter slides within a diagnosed SAD group revealed that a sample size of 23 participants per group would have 80% power to detect an effect size of 0.75 (Rohan et al., 2003). When comparing the same SAD group to a nonseasonal, nondepressed control group and examining POMS change scores to bright light slides and collapsing across the seasons, analyses revealed that 10 participants per group would provide 80% power to detect an effect size of 1.18. In a different study, examining POMS change scores to bright light slides and collapsing across the seasons, participants with S-SAD were compared to normal controls. This analysis revealed that a sample size of 18 participants per group would have 80% power to detect an effect size of 0.85 (Rohan et al., 2004).

Finally, a review of numerous surface facial EMG studies revealed a diverse sample size requirement based on the use of different types of stimuli and varying methodological procedures such as generation of happy and sad “imagery” (e.g., Greden

et al., 1984; Schwartz et al., 1976b), visual inspection of self-referent statements with elated, depressed, and neutral content (Sirota & Schwartz, 1982), and exposure to happy and angry facial expressions (e.g., Dimberg, 1982; Dimberg & Lundquist, 1990). Given that most EMG studies reviewed were somewhat dated and did not routinely report means and standard deviations, there were few studies on which appropriate power analyses could be conducted. However, two studies that found significant differences in EMG corrugator activity when comparing depressed and nondepressed participants reported cell sizes of 12 participants (Schwartz et al., 1976a, Schwartz et al., 1976b).

When examining corrugator reactivity in a study comparing dysphoric and nondysphoric college students, significant differences were found between the groups with cell sizes of 21 participants. In one study examining corrugator muscle activity in response to personally-relevant sad imagery and comparing normal controls to endogenously depressed participants, a sample size of 25 participants per group would have 80% power to detect an effect size of 0.73 (Greden et al., 1986). Given these data, we postulated a similar effect size as SCR, however, we did not correct for multiple dependent variables. Because we were interested in finding differences between groups of at least 0.05, anything smaller may not be clinically or theoretically meaningful.

## RESULTS

### *Participant Characteristics*

Analyses included data from 48 participants: 24 SAD and 24 controls. The groups did not differ on age, gender, race, marital status, or education; all *p* values *ns* (See Table 4). Generally, the participant pool was made up of individuals who were middle-aged (*M* = 41.71, *SD* = 12.12), female (91.7%), Caucasian (70.8%), married

(56.3%), with a college degree (75.0%), and employed (93.7%). In addition, there were no significant differences between SAD and control groups during baseline on SCL, $t$ (46) = 1.43, $p$ = .16 (SAD: $M$ = 2.64, $SD$ = 1.52; Control: $M$ = 1.99, $SD$ = 1.59); EMG corrugator, $t$ (46) = - .068, $p$ = .95 (SAD: $M$ = 7.52, $SD$ = 3.30; Control: $M$ = 7.59, $SD$ = 4.00); lower zygomatic, $t$ (46) = 1.74, $p$ = .088 (SAD: $M$ = 5.12, $SD$ = 3.61; Control: $M$ = 3.72, $SD$ = 1.54), or upper zygomatic $t$ (46) = 1.88, $p$ = .067 (SAD: $M$ = 5.03, $SD$ = 4.72; Control: $M$ = 3.06, $SD$ = 2.02). The SAD and control groups did not differ on the VVIQ, $t$ (46) = .87, $p$ = .39 (SAD: $M$ = 33.00, $SD$ = 9.05; Control: $M$ = 30.54, $SD$ = 10.45), suggesting that the groups were well-matched on imagery ability.

Descriptively, there were no differences between SAD and control female participants on current menstrual cycle phase. Specifically, the groups had comparable percentages in the premenstrual (late phase luteal) phase: SAD ($n$ = 2, 8%), control ($n$ = 2, 8%). Finally, when matching participants' psychophysiological assessments across time-of-day to control for differences in circadian rhythms, a greater percentage of SAD participants were assessed in the evening, whereas, a greater percentage of the control group was assessed in the middle of the day. The assessments took place from 7:00 - 10:00 am (29.2 % SAD, 20.8% control), 12:00 - 3:00 pm (16.7% SAD, 45.8% control), and 5:00 - 8:00pm (54.2% SAD, 33.3% control). However, time-of-day and menstrual cycle phase did not appear to be significantly related to the outcome measures, in that the same number of SAD and control participants in the late phase luteal phase of the menstrual cycle were assessed between 12:00 – 3:00 pm (i.e., 2) and between 5:00 – 8:00pm (i.e., 2).

***Preliminary Data Inspection***

Prior to analyses, distributions, means, standard error of the means, and standard deviations were inspected for each of the psychophysiological measures. Skin conductance measures (SCR frequency and magnitude) approximated a normal distribution with reasonable between- and within-subjects variability. Concerning EMG results, due to extreme variability both between- and within-subjects on the peak EMG measure, it was determined that mean EMG represents a better dependent measure because it had less variance and was approximately normally distributed. Therefore, mean EMG will serve as the primary outcome variable of interest. Inspection of the data also revealed stronger differences during initial slide presentation within each block of slides.

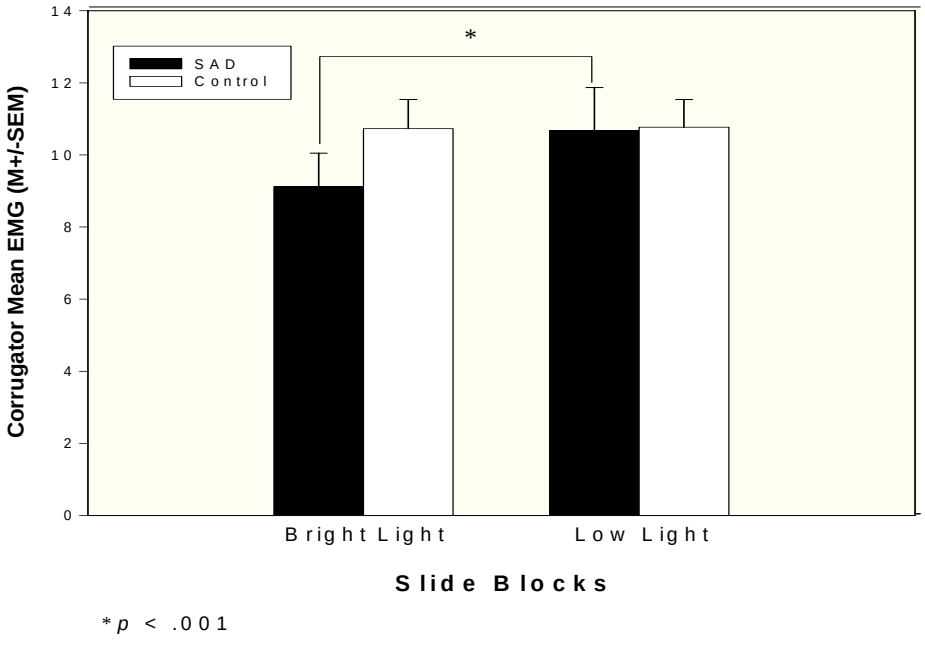
***Hypothesis 1: Corrugator Supercilii Activity in Response to Low Light and Winter Stimuli***

***Mean Corrugator EMG.*** The EMG dependent measures for the SAD and control groups at slide presentation are shown in Table 5. A 2 (participant group; SAD, control) X 2 (light intensity; bright light, low light) X 3 (season; summer, fall, winter) repeated measures ANOVA on mean EMG corrugator supercilii activity during slide presentation did not reveal a Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = .50, $p$ = .61, $\eta^2$ = .022, *ns*. However, a significant Group X Light interaction was found, Wilks' Lambda $F$ (1, 46) = 6.75, $p$ = .013, $\eta^2$ = .13. Main effect analyses revealed a main effect of light, collapsing across seasons, within the SAD group, Wilks' Lambda $F$ (1, 46) = 14.20, $p < .001$, $\eta^2$ = .24, whereby SAD participants emitted increased corrugator activity (i.e., brow-pursing, correlated with a frown response) during exposure to low

light slides ($M$ = 10.67) as compared to bright light slides ($M$ = 9.12). Although there were no significant group differences, descriptively, the overall pattern of mean corrugator EMG to light-relevant stimuli for SAD versus control participants suggests that the main hypothesis was not perfectly confirmed. That is, SAD participants actually evidenced decreased mean corrugator EMG upon exposure to bright light slides relative to any other group/light intensity cell. This pattern may be representative of exaggerated positive reactions and/or reduced negative reactions to bright light stimuli as opposed to increased negative reactions to low light stimuli. Therefore, the present results suggest that bright light stimuli may be more influential on facial expression of emotion than low light stimuli in SAD (See Figure 6). There was no group main effect at either low light or bright light intensity, collapsing across season.

***Figure 6.***

**Mean EMG Corrugator Supercilii Activity During Slide Presentation, Collapsing Across Season.**

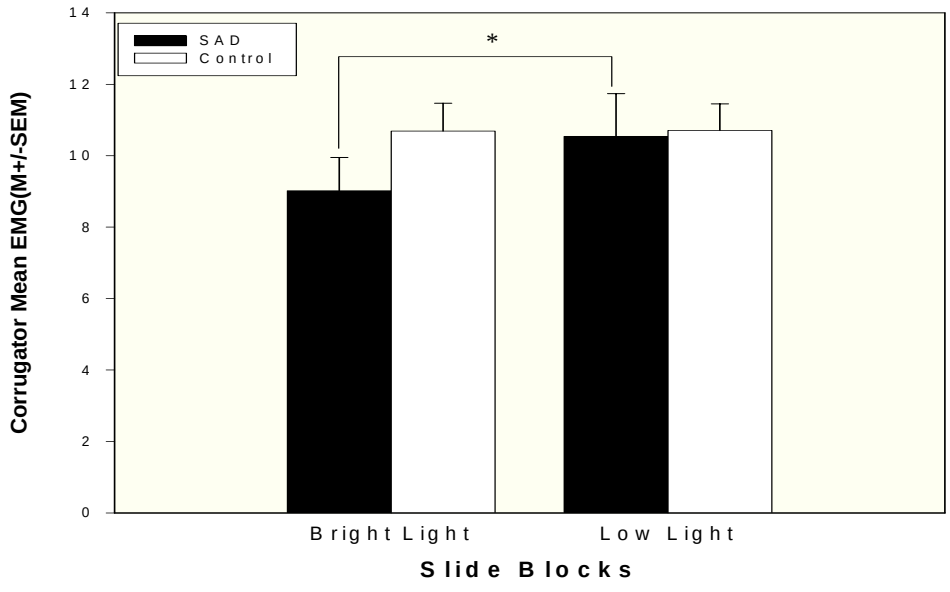



*$p$ < .001

When examining mean EMG corrugator activity during the interval following slide offset (See Table 6), no Group X Light X Season interaction was found, Wilks' Lambda $F$ (2, 45) = .048, $p$ = .95, $\eta^2$ = .002. However, a significant Group X Light interaction was found, Wilks' Lambda $F$ (1, 46) = 6.63, $p$ = .013, $\eta^2$ = .13. Main effect analyses revealed a main effect of light, collapsing across seasons, within the SAD group (See Figure 6.1), Wilks' Lambda $F$ (1, 46) = 13.75, $p$ = .001, $\eta^2$ = .23, with SAD participants emitting increased corrugator activity after exposure to low light slides ($M$ = 10.54) as compared to bright light slides ($M$ = 9.02). Visual inspection of the overall pattern of results suggests the same pattern that was observed during slide presentation. Specifically, SAD participants appeared to demonstrate reduced mean corrugator EMG to bright light slides relative to low light slides during the interval following slide offset.

***Figure 6.1***

**Mean EMG Corrugator Supercilii Activity During Slide Offset, Collapsing Across Season.**




* $p$ = .001

***Peak Corrugator EMG.*** 2 (participant group; SAD, control) X 2 (light intensity; bright, low light) X 3 (season; summer, fall, winter) repeated measures ANOVAs on peak EMG corrugator activity during slide presentation (See Table 7), Wilks' Lambda $F$ (2, 45) = 1.88, $p$ = .17, $\eta^2$ = .077, and during slide offset (See Table 8), Wilks' Lambda $F$ (2, 45) = .47, $p$ = .63, $\eta^2$ = .020, revealed no significant Group X Light X Season interactions. Further, there were no significant 2-way interactions or main effects for peak EMG corrugator activity.

### *Hypothesis 2: Zygomaticus Major Activity in Response to Bright Light and Summer Stimuli*

#### *Lower Zygomaticus Major - Channel 3*

***Mean Lower Zygomaticus EMG.*** A 2 (participant group; SAD, controls) X 2 (light intensity; bright light, low light) X 3 (season; summer, fall, winter) repeated measures ANOVA on mean EMG lower zygomatic activity during slide presentation did not reveal a significant Group X Light X Season interaction (See Table 9), Wilks' Lambda $F$ (2, 45) = 1.45, $p$ = .25, $\eta^2$ = .061. The 3-way interaction was also *ns* for lower zygomatic mean EMG during slide offset (See Table 10), Wilks' Lambda $F$ (2, 36) = .004, $p$ = .10, $\eta^2$ < .001. In addition, the ANOVAs revealed no significant 2-way interactions or main effects during the slide presentation or offset intervals for mean EMG lower zygomatic activity.

***Peak Lower Zygomaticus EMG.*** When examining peak EMG lower zygomatic activity during slide presentation (See Table 11), the Group X Light X Season interaction was *ns*, Wilks' Lambda $F$ (2, 45) = 1.91, $p$ = .16, $\eta^2$ = .078. Likewise, when examining peak EMG lower zygomatic activity during slide offset (See Table 12), no significant Group X Light X Season interaction was found, Wilks' Lambda $F$ (2, 45) = .47, $p$ = .63,

$\eta^2$ = .020. There were also no significant 2-way interactions or main effects revealed on peak EMG lower zygomatic activity during the slide presentation or offset intervals.

***Upper Zygomaticus Major - Channel 4***

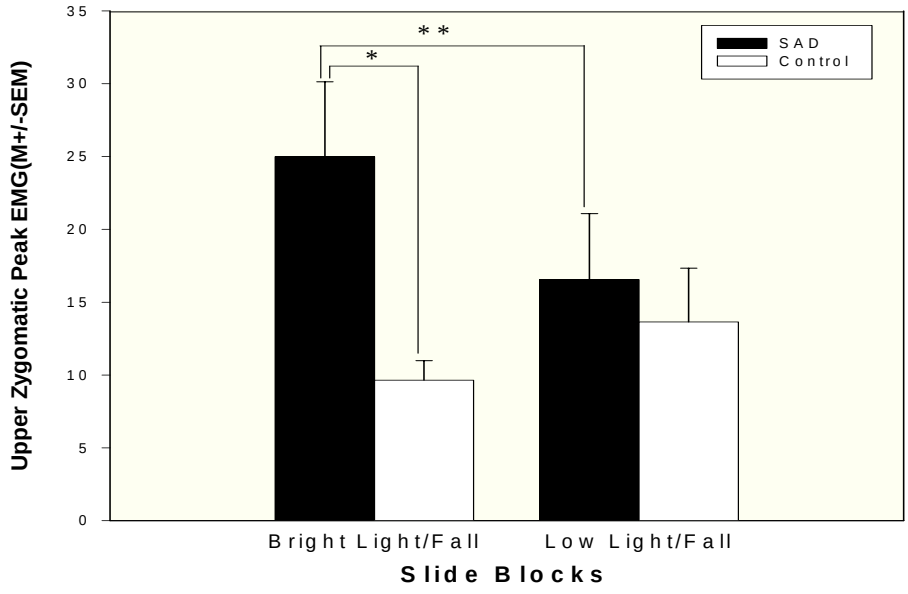
***Mean Upper Zygomaticus EMG.*** EMG dependent measures for SAD and control groups at slide presentation and offset are presented in Tables 13 and 14. A 2 (participant group; SAD, controls) X 2 (light intensity; bright light, low light) X 3 (season; summer, fall, winter) repeated measures ANOVA on mean EMG upper zygomatic activity during slide presentation revealed no significant Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = 2.26, $p$ = .12, $\eta^2$ = .091. The 3-way interaction was also *ns* for mean EMG upper zygomatic activity during slide offset, Wilks' Lambda $F$ (2, 45) = .89, $p$ = .42, $\eta^2$ = .038. In addition, no significant 2-way interactions or main effects were found for mean EMG upper zygomatic activity during either the slide presentation or offset intervals.

***Peak Upper Zygomaticus EMG.*** Dependant measures for upper zygomatic peak EMG slide onset for SAD and control groups are shown in Table 15. When examining peak EMG upper zygomatic activity during slide presentation, a 2 (participant group; SAD, control) X 2 (light intensity; bright light, low light) X 3 (season; summer, fall, winter) repeated measures ANOVA revealed a significant Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = 3.74, $p$ = .031, $\eta^2$ = .14. Simple interaction effect analyses were conducted to explore the Group X Light interaction within each season. Results revealed a nonsignificant Group X Light interaction within summer slides, $F$ (1, 46) = .35, $p$ = .56, $\eta^2$ = .008 and a nonsignificant Group X Light interaction within winter slides, $F$ (1, 46) = 1.35, $p$ = .25, $\eta^2$ = .028. However, a significant Group X Light

interaction within fall season was found, $F$ (1, 46) = 9.32, $p$ = .004, $\eta^2$ = .17. Simple effect analyses revealed significant group differences in peak upper zygomatic activity during presentation of bright light/fall slides, $F$ (1, 46) = 8.34, $p$ = .006, $\eta^2$ = .15, where SAD participants ($M$ = 24.99) emitted greater zygomatic activity (i.e., smile response) as compared to controls ($M$ = 9.64). In addition, simple effect analyses demonstrated a main effect of light within fall season slides for SAD participants (See Figure 6.2), $F$ (1, 46) = 8.60, $p$ = .005, $\eta^2$ = .16, whereby the SAD group evidenced greater peak upper zygomatic activity to bright light/fall slides ($M$ = 24.99) as compared to low light/fall slides ($M$ = 16.55).

***Figure 6.2***

**Peak EMG Upper Zygomaticus Major Activity During Slide Presentation, Collapsing Across Season.**




**p = .006. **p =.005.*

When examining peak EMG upper zygomatic activity during slide offset (See Table 16), the Group X Light X Season interaction was *ns*, Wilks' Lambda $F$ (2, 45)

= .10, $p$ = .90, $\eta^2$ = .004. There were also no significant 2-way interactions or main effects revealed on peak EMG upper zygomatic activity during either slide presentation or offset.

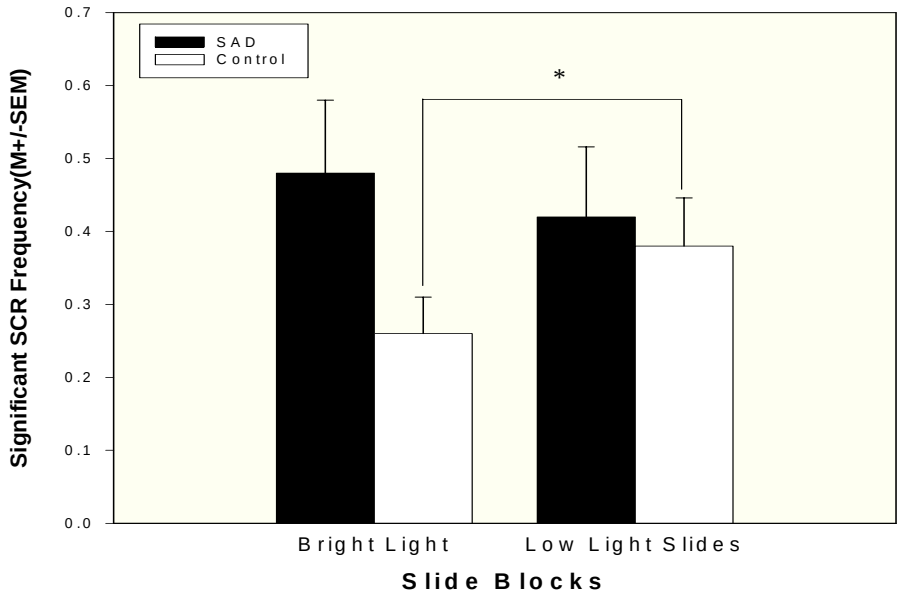
***Hypothesis 3: Significant Skin Conductance Responses and Skin Conductance Response Magnitude to Low Light and Winter Stimuli***

***Significant Skin Conductance Response (SCR) Frequency***

Dependent measures for SCR Frequency at slide onset can be found in Table 17 for SAD and controls. The ANOVA on skin conductance response (SCR) frequency during slide presentation revealed no significant Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = .27, $p$ = .77, $\eta^2$ = .012. However, a significant Group X Light interaction was found, Wilks' Lambda $F$ (1, 46) = 8.24, $p$ = .006, $\eta^2$ = .15. Main effect analyses revealed a main effect of light (See Figure 6.3), collapsing across seasons, within the control group, Wilks' Lambda $F$ (1, 46) = 6.99, $p$ = .011, $\eta^2$ = .13, whereby control participants evidenced increased significant SCR frequency during low light slides ($M$ = .38) as compared to bright light slides ($M$ = .27).

*Figure 6.3*

**Significant Skin Conductance Response (SCR) Frequency During Slide Presentation, Collapsing Across Season.**




*$p$ = .011.

Examining significant SCR frequency during slide offset (See Table 18) did not reveal a Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = .038, $p$ = .96, $\eta^2$ = .002. Further, there were no significant 2-way interactions or main effects.

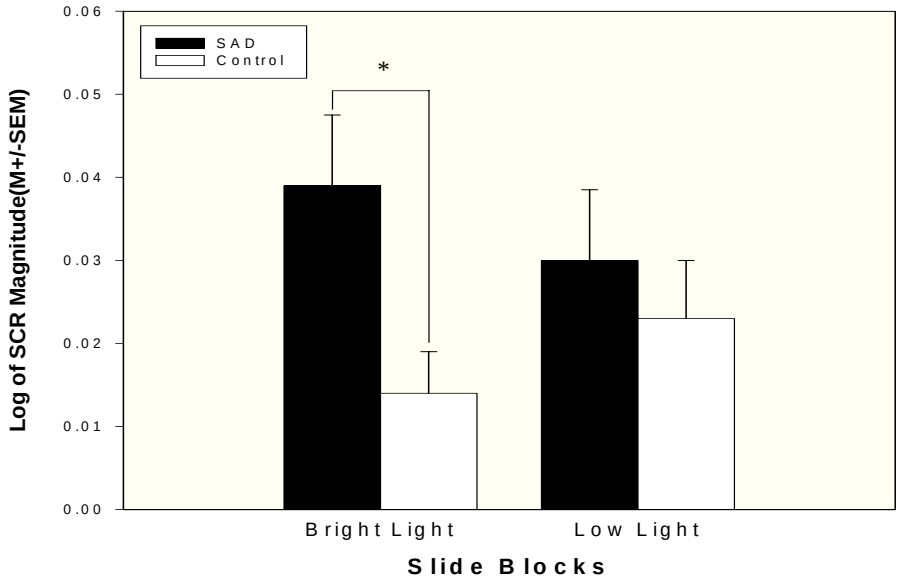
***Skin Conductance Response (SCR) Magnitude***

SAD and control dependent measures for SCR magnitude during slide presentation are presented in Table 19. A 2 (participant group; SAD, control) X 2 (light intensity; bright light, low light) X 3 (season; summer, fall, winter) repeated measures ANOVA on the skin conductance response (SCR) magnitude during slide presentation revealed no Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = 1.93, $p$ = .16, $\eta^2$ = .079. However, a significant Group X Light interaction was found, Wilks' Lambda $F$ (1, 46) = 6.67, $p$ = .013, $\eta^2$ = .13. Main effect analyses revealed a main effect of group, collapsing across seasons, within light stimuli (See Figure 6.4), Wilks' Lambda

$F$ (1, 46) = 6.36, $p$ = .015, $\eta^2$ = .12, whereby SAD participants ($M$ = .04) emitted greater SCR magnitude during bright light slides as compared to controls ($M$ = .01).

***Figure 6.4***

**Skin Conductance Response (SCR) Magnitude During Slide Presentation, Collapsing Across Season.**




*$p$ = .015.

No Group X Light X Season interaction was revealed for SCR magnitude during slide offset (See Table 20), Wilks' Lambda $F$ (2, 45) = .087, $p$ = .92, $\eta^2$ = .004. Further, there were no significant two-way interactions or main effects.

***Hypothesis 4: Depression-Dejection Subscale Scores on the Profile of Mood States and Lower Perceived Pleasantness Ratings for Low Light and Winter Stimuli***

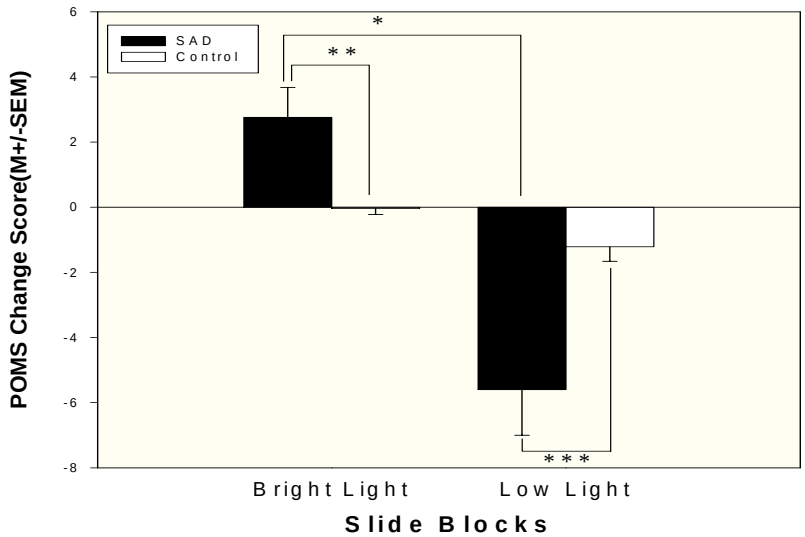
***POMS Depression-Dejection Subscale Scores***

Table 21 displays POMS Depression-Dejection subscale change scores from baseline for SAD and control groups. A 2 (participant group; SAD, control) X 2 (light intensity; bright light, low light) X 3 (season; summer, fall, winter) repeated measures ANOVA on Profile of Mood States (POMS) Depression-Dejection subscale change

scores from baseline revealed no significant Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = 1.92, $p$ = .16, $\eta^2$ = .079. However, a significant Group X Light interaction was found, Wilks' Lambda $F$ (1, 46) = 29.75, $p$ < .001, $\eta^2$ = .39. According to Cohen (1988), this is considered a large effect size. In addition, a Group X Season interaction was found, Wilks' Lambda $F$ (2, 45) = 4.98, $p$ = .011, $\eta^2$ = .18. Main effect analyses revealed a main effect of light, collapsing across season, within the SAD group (See Figure 6.5), Wilks' Lambda $F$ (1, 46) = 80.36, $p$ < .001, $\eta^2$ = .64 (a very large effect size; Cohen, 1988), whereby SAD participants reported greater exacerbation of baseline depressed mood after viewing the low light slides ($M$ = -5.60) than subsequent to the bright light slides ($M$ = 2.76).

***Figure 6.5***

**POMS Depression-Dejection Subscale Change Scores From Baseline, Collapsing Across Season.**




*Note*. Change score = Baseline POMS - Stimulus block POMS. Negative change scores inidcate an exacerbation of baseline depressed mood. Positive change scores indicate an improvement above baseline depressed mood. *$p$ < .001. **$p$ = .005. ***$p$ = .005.

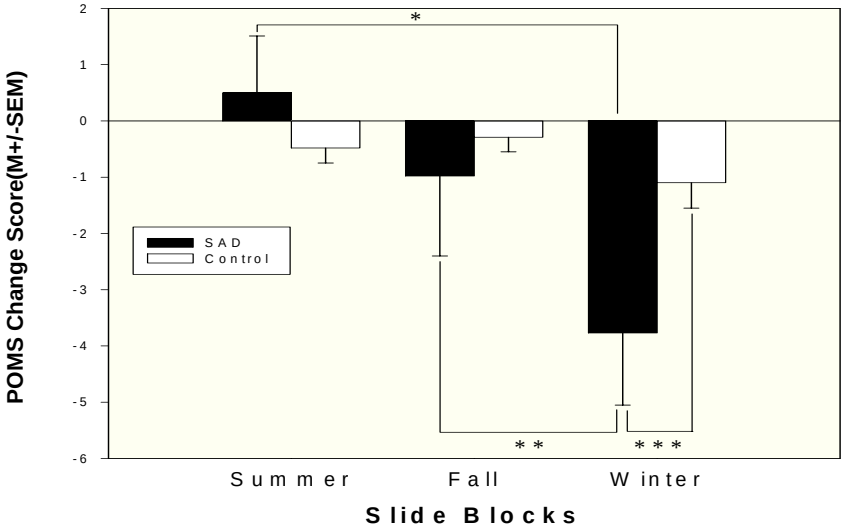
In addition, Main effect analyses revealed a main effect of group, collapsing across season, within the bright light slides, Wilks' Lambda $F$ (1, 46) = 8.92, $p$ = .005,

$\eta^2$ = .16, and within the low light slides, Wilks' Lambda $F$ (1, 46) = 8.90, $p$ = .005, $\eta^2$ = .16. Relative to controls ($M$ = -.04), SAD participants reported a greater improvement in depressed mood after viewing bright light slides ($M$ = 2.76). In addition, SAD participants reported a greater exacerbation of depressed mood subsequent to the low light slides ($M$ = -5.60) as compared to controls ($M$ = -1.21).

Further Main effect analyses revealed a main effect of season, collapsing across light, within the SAD group (See Figure 6.6), Wilks' Lambda $F$ (2, 45) = 14.12, $p$ < .001, $\eta^2$ = .39. SAD participants reported greater exacerbation of depressed mood after viewing winter slides ($M$ = -3.77) as compared to summer slides ($M$ = .50), $t$ (23) = -3.83, $p$ = .001, and greater exacerbation of depressed mood subsequent to winter slides ($M$ = -3.77) as compared to fall slides ($M$ = -.98), $t$ (23) = -2.10, $p$ = .047).

***Figure 6.6***

**POMS Depression-Dejection Subscale Change Scores From Baseline, Collapsing Across Light.**




*Note*. Change score = Baseline POMS - Stimulus block POMS. Negative change scores inidcate an exacerbation of baseline depressed mood. Positive change scores indicate an improvement above baseline depressed mood. *$p$ = .001. **$p$ = .047. ***$p$ = .055.

In addition, Main effect analyses revealed a main effect of group, collapsing across light, on winter slides, $F$ (1, 46) = 3.88, $p$ = .055, $\eta^2$ = .08, SAD participants ($M$ = -3.77) reporting greater exacerbation of baseline depressed mood after winter slides than controls ($M$ = -1.11).

***Perceived Pleasantness Ratings***

Pleasantness ratings are presented in Table 22. When examining pleasantness ratings after slide presentation, a 2 (participant group; SAD, control) X 2 (light intensity; bright light, low light) X 3 (season; summer, fall, winter) repeated measures ANOVA revealed a significant Group X Light X Season interaction, Wilks' Lambda $F$ (2, 45) = 3.06, $p$ = .057, $\eta^2$ = .12. Simple interaction effect analyses were conducted to explore the Group X Light interaction within each season. Results revealed no Group X Light interaction within winter slides, $F$ (1, 46) = .60, $p$ = .44, $\eta^2$ = .013. However, a significant Group X Light interaction within summer slides, $F$ (1, 46) = 8.96, $p$ = .004, $\eta^2$ = .16, and a significant Group X Light interaction within fall slides were found, $F$ (1, 46) = 14.10, $p$ < .001, $\eta^2$ = .24. Main effect analyses revealed significant group differences in perceived pleasantness ratings after viewing low light/summer slides, $F$ (1, 46) = 11.60, $p$ = .001, $\eta^2$ = .20, where SAD participants ($M$ = 2.38) perceived a lower degree of pleasantness as compared to controls ($M$ = 3.58). Additional main effect analyses revealed significant group differences in perceived pleasantness ratings after viewing low light/fall slides, $F$ (1, 46) = 30.71, $p$ < .001, $\eta^2$ = .40, where SAD participants ($M$ = 2.88) perceived a lower degree of pleasantness as compared to controls ($M$ = 4.59).

Further simple effect analyses revealed a light main effect in both the SAD,

$F$ (1, 46) = 117.39, $p < .001$, $\eta^2 = .72$, and control groups, $F$ (1, 46) = 43.57, $p < .001$, $\eta^2 = .49$, for summer slides. Specifically, SAD reported lower perceived pleasantness for low light/summer slides ($M = 2.38$) as compared to bright light/summer slides ($M = 6.00$) and controls showed the same pattern: low light/summer slides ($M = 3.58$) were rated as less pleasant than bright light/summer slides ($M = 5.79$). Simple effect analyses also revealed a light main effect for both the SAD, $F$ (1, 46) = 98.27, $p < .001$, $\eta^2 = .68$, and control groups, $F$ (1, 46) = 21.18, $p < .001$, $\eta^2 = .32$, for fall slides. Specifically, SAD participants perceived low light/fall slides as less pleasant ($M = 2.88$) than bright light/fall slides ($M = 5.21$) and controls showed the same pattern: low light/fall slides ($M = 4.59$) were rated as less pleasant in comparison to bright light/fall slides ($M = 5.67$).

***Ancillary Analyses***

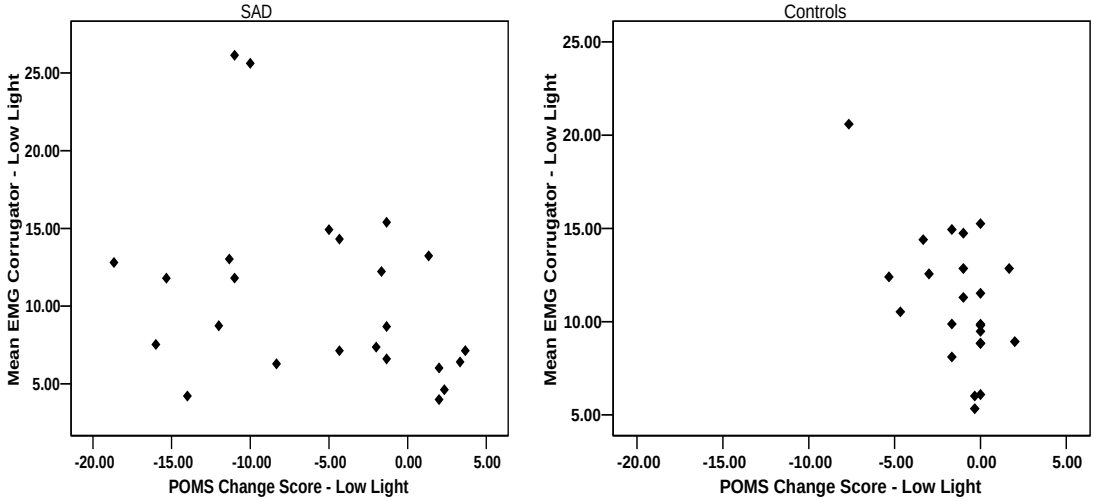
James (1884) suggested that facial expression of emotion is associated with the experiencing of more intense emotions. This concept may have applicability to the present study. For example, low light cues associated with exacerbated depressed mood may result in increased POMS Depression-Dejection subscale scores as well as increased corrugator reactivity (i.e., brow-pursing). The resultant heightened intensity of negative mood could contribute to further increases in self-reported depressed mood state and further increases in corrugator response, an example of proprioception.

Consistent with the concept of proprioception, additional analyses were performed to explore whether the magnitude of significant mean EMG corrugator responses were related to the degree of self-reported depression change on the POMS. First, Pearson correlations were computed to determine whether POMS change scores following low light stimuli correlated with mean EMG corrugator response to low light

stimuli. For the SAD group, correlations between POMS Depression-Dejection change scores and mean corrugator EMG activity to low light stimuli were nonsignificant, $r = -.32$, $p = .13$. However, for the control group, POMS change scores and mean EMG corrugator activity to low light stimuli correlated significantly, $r = -.55$, $p = .006$ (See Figure 6.7).

***Figure 6.7***

**Mean EMG Corrugator Supercilii and POMS Depression-Dejection Subscale Change Scores for Low Light Stimuli in SAD and Control Participants.**

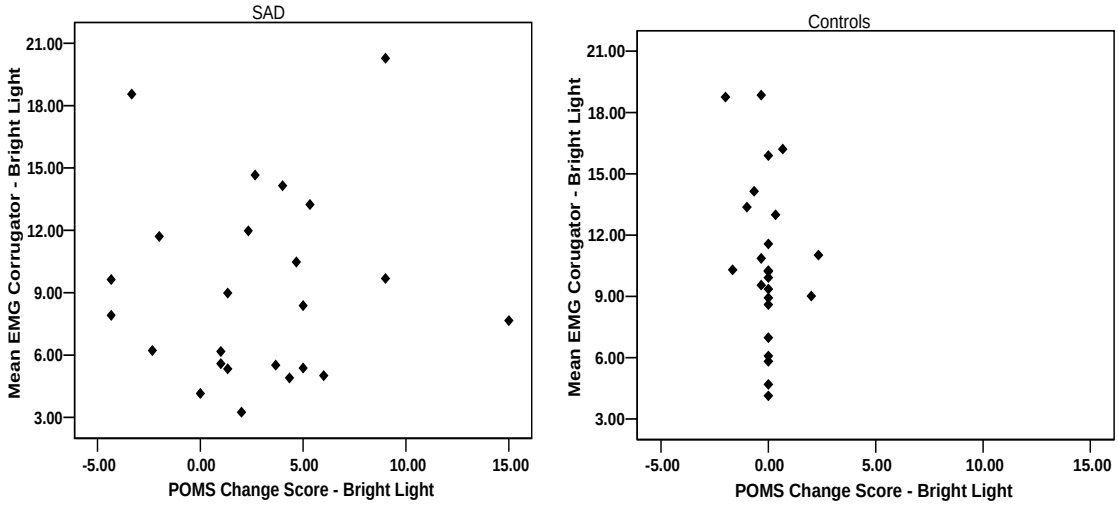



In addition, correlations between POMS Depression-Dejection change scores and mean corrugator EMG activity to bright light stimuli were nonsignificant for both the SAD group, $r = .053$, $p = .81$, and the control group, $r = -.25$, $p = .23$ (See Figure 6.8). In summary, in reaction to low light slides, controls demonstrated better concordance between facial affect (i.e., brow-pursing) and self-reported depressed mood than SAD participants. However, in reaction to bright light slides, neither SAD nor control participants demonstrated significant agreement between facial affect (i.e., brow-

pursing), and self-reported depressed mood, suggesting that proprioception was not operative in this study.

***Figure 6.8***

**Mean EMG Corrugator Supercilii and POMS Depression-Dejection Subscale Change Scores for Bright Light Stimuli in SAD and Control Participants.**




***Menstrual Cycle Phase.*** A post-hoc inspection of the data was conducted to determine whether current menstrual cycle phase, specifically the premenstrual phase, may have confounded significant findings for mean EMG corrugator reactivity and POMS Depression-Dejection subscale change scores. Although these exploratory analyses are under-powered, descriptive results suggest that women in the premenstrual phase did not differ from other women on mean EMG corrugator reactivity during slide presentation or offset or POMS change scores. Consequently, current menstrual cycle phase does not appear to be a confound to findings obtained on mean EMG corrugator activity or the POMS (See Table 23).

***Time-of Day.*** Similarly, post-hoc analyses were performed to explore whether time-of-day may have confounded the findings. Post-hoc inspection of the data revealed

that participants assessed 7-10 am, 12-3 pm, and 5-8 pm did not differ on mean EMG corrugator activity or POMS Depression-Dejection subscale change scores across the blocks of slides. Therefore, the time-of-day when participants were assessed does not appear to have been a confound in this study (See Table 24).

## DISCUSSION

To our knowledge, this study is the first to explore facial muscle patterning responses, measured through surface facial electromyography (EMG), in SAD. In particular, this study examined whether individuals with diagnosed SAD evidence increased EMG responding to environmental stimuli that differ by light intensity and season relative to nonseasonal, nondepressed controls. In addition to investigation of emotion-specific psychophysiological responding, another goal of the study was to determine whether individuals with SAD evidence increased general sympathetic arousal [i.e., significant skin conductance response (SCR) frequency and greater SCR magnitude] than controls.

In general, this study found evidence of heightened EMG reactivity in SAD as compared to controls. The most striking evidence of emotion-specific psychophysiological reactivity was found in the corrugator muscle. A significant body of research has shown that corrugator activity is a reliable measure of covert emotion-specific reactivity to negative or unhappy imagery (e.g., Cacioppo et al., 1988; Schwartz et al., 1980). In the present study, SAD participants differed in their mean EMG corrugator reactions to bright versus low light stimuli. In contrast, controls did not differ in corrugator responses to low versus bright light stimuli. The main hypothesis concerning corrugator response was not perfectly supported in that individuals with SAD

did not necessarily demonstrate higher mean corrugator reactivity (i.e., brow pursing associated with a frown response) while viewing low light slides as compared to bright light slides; rather, they appeared to emit decreased mean corrugator reactivity to the bright light slides. Even though the groups did not differ on mean EMG corrugator activity, this assertion is based on the descriptive pattern observed across SAD and control participants to light-relevant stimuli. As such, the bright light slides appear to have a mood-enhancing effect on participants with SAD. This may indicate a less negative and/or a more positive reaction to bright light than to low light stimuli in SAD. These results support the hypothesis that a dark, dreary sky may be a conditioned stimulus for depressive affect in SAD. Similarly, bright, sunny skies may be a conditioned stimulus for alleviation of depressed mood in SAD. Corrugator results reported here are also consistent with literature suggesting that environmental stimuli representative of low light conditions may be considered unpleasant and stimuli representing bright light may be pleasant in SAD (Rohan et al., 2003). These findings are consistent with literature suggesting that EMG response is spontaneous and happens within a few milliseconds of stimulus onset (Cacioppo et al., 2000; Eckman et al., 1972).

What may be even more interesting in the present study is that the SAD group continued to show the same reduced mean corrugator response pattern to bright light slides as compared to low light slides during the 10-second interval following slide offset. Therefore, in SAD participants, the reduced brow pursing response persisted beyond the actual exposure to a bright light stimulus. Possible mediators of this lingering negative affective reaction include negative cognitions about dark, dreary weather, either automatic or intentional, that drive a sustained corrugator response. According to

response styles theory (Nolen-Hoeksema, 1987), rumination is triggered by a negative event. It may be that low light stimuli trigger a cascade of negative cognitive ruminations about dreary weather, its intrinsic meaning, and its personal implications that cause brow-pursing to persist over time in individuals with SAD. This is consistent with Rohan et al.'s (2004) hypothesis that SAD may involve core beliefs related to light availability. Alternatively, it is worth considering that the 10-second slide onset and 10-second slide offset periods are of insufficient duration for psychophysiological responding to return to natural baseline (Roth, personal communication, 2003). However, the latter explanation seems unlikely given that the sustained contraction of corrugator muscle fibers was unique to the SAD group and was only observed for low light slides.

In addition to psychophysiological responding, Profile of Mood States Depression-Dejection subscale (POMS; McNair et al., 1971) results indicate that individuals with SAD react with a negative affect to low light stimuli. Relative to controls, SAD participants reported improvements in depressed mood from baseline to exposure to bright light stimuli and exacerbated depressed mood after exposure to low light slides. In addition, SAD participants reported greater increases in depressed mood over their baseline level subsequent to viewing winter slides, relative to control participants. Among individuals with SAD, the rise in transient depressive affect was specific to winter stimuli and did not manifest with fall or summer stimuli. Therefore, subjectively, individuals with SAD reported that the low light and winter content slides made them "feel" more depressed, and that bright light stimuli made them feel less depressed.

One possible explanation for the convergence of the findings for self-reported depressed mood and corrugator EMG responding outcome data to low light stimuli comes from Rohan et al.'s (2003), extension of Lewinsohn's (1985) sensitivity to aversive events hypothesis. That is, if depressive episodes are repeatedly paired with decreased light availability in SAD, environmental cues signaling low light may become conditioned stimuli for depressive affect and/or a negative cognitive interpretation of such cues. This conditioning and overgeneralization may result in a hypersensitivity (i.e., increased physiological responding) to low light stimuli specifically on the corrugator, which taps into negative emotion. In addition, Lewinsohn (1985) proposed that environmental and situational factors represent the primary triggers of the depressive response (e.g., exposure to low light in SAD), and that cognitions serve as moderators to the effects of the environment. To the extent that negative emotional reactions are critical to individual functioning, a self-focus process initiates. This heightened self-focus, in combination with dysphoric mood, may initiate a cascade of other negative cognitions and behaviors that serve to maintain negative affect, consistent with our findings on POMS and EMG corrugator activity.

Results from the EMG literature examining depressed, nondepressed, dysphoric, and nondysphoric samples indicate that the corrugator may be a reliable psychophysiological marker of negative emotion-specific reactivity. Overall, studies have consistently shown heightened corrugator EMG response to unpleasant or negative stimuli, in both clinical and depressed samples (Oliveau & Willmuth, 1979; Schwartz et al., 1976a, 1976b). Therefore, based on corrugator and mood findings, it seems reasonable to conclude that stimuli depicting low light weather conditions are at least

unpleasant, and perhaps even aversive, to individuals with SAD. This conclusion is also supported by the present study's perceived pleasantness ratings, whereby SAD participants reported a lower perception of pleasantness on both low light/summer and low light/fall slides as compared to controls.

These results are also consistent with several studies that show convergence between self-reported mood state and indices of psychophysiological arousal in depression (e.g., Rohan et al., 2004; Schwartz et al., 1980; Sigmon et al., 2002; Teasdale & Bancroft, 1977). As Winkielman and Cacioppo (2001) discuss, the simultaneous collection of both self-reported mood states and psychophysiological responses, allow examination of implicit or nonverbal processes, and may assist researchers in developing and testing theoretical models. Psychophysiological measures may have greater sensitivity to detect subtle changes in arousal as compared to more conventional self-report measures (Winkielman & Cacioppo, 2001). Specifically, a "triangulation" approach may help to determine if conscious or unconscious psychological processes are expressing themselves internally through a change in affect and/or externally through heightened psychophysiological responses.

However, results in the present study suggest that this may be true only in very specific domains. For example, counter to the hypothesis that individuals with SAD would evidence increased SCR frequency and SCR magnitude to low light/winter slides (presumed to be unpleasant), only control participants demonstrated more frequent SCRs to low light slides than bright light slides. Equally unexpected, SAD participants reacted with heightened SCR magnitude to bright light slides, relative to low light slides. Perhaps SCR and SCR magnitude do not posses appropriate specificity for the task.

In a prior study, happy and angry facial expressions evoked increased EMG change scores from stimulus onset (second by second during stimulus presentation) in the expected direction (Dimberg, 1982). Results demonstrated increased corrugator response to angry as compared to happy facial expressions, and increased zygomatic region activity to happy, as opposed to angry faces. Additionally, this study found decreased significant SCRs as a function of time for both angry and happy stimuli, which may represent an indicator of an orienting response (Graham, 1973; Sokolov, 1963). Further, SCR response did not differentiate between happy and angry stimuli, a finding that may be relevant to the present study. In our investigation, SCR also evidenced a similar reactivity pattern over time whereby there was a significant main effect of time on significant SCR response over each block of slides and a similar main effect of time across slide blocks on SCR magnitude at both slide onset and slide offset. The fact that skin conductance findings were inconsistent with other dependent measures that yielded a consistent pattern (i.e., corrugator EMG, POMS depression-dejection scores, and perceived pleasantness ratings), could be an artifact of an orienting response, or an example of directional fractionation, in the case of POMS and SCR.

The concept of directional fractionation is based on three separate domains of arousal: behavioral, autonomic, and cortical (Lacey, 1967). Because arousal is not a simple, unidimensional continuum, but a complex interrelated system, individuals may not respond in the same manner across systems. Consequently, even within a given individual, autonomic responding may not be uniform across the various arousal dimensions. In the behavioral domain, SAD participants appear to be reacting to the low light stimuli with increased negative mood (as assessed by the POMS) and report that

they see these stimuli as unpleasant. In contrast, SCR and SCR magnitude responses, considered autonomic indices of arousal, did not show this same pattern and, in fact, appeared to move in the opposite direction. Consequently, both directional fractionation and lack of specificity within skin conductance response may help explain the discrepant results between SCR and SCR magnitude with the convergent results demonstrated by POMS, perceived pleasantness ratings, and EMG corrugator (i.e., a somatic response). Although research findings on the correlation between autonomic and somatic domain response is sparse, studies have shown that there may also be differential response patterns between these two domains (Dimberg, 1982).

It is important to note that skin conductance results were not consistent with findings from previous SAD studies. Two prior studies employed light-relevant stimuli (i.e., slides of outdoor scenes with varying light intensity; Rohan et al., 2003; 2004) and another employed season-relevant stimuli (i.e., a video depicting summer and winter scenes in Maine; Sigmon et al., 2002). Rohan et al. (2004) found that women with S-SAD demonstrated greater SCR magnitude in reaction to low light stimuli than nondepressed controls, regardless of season assessment. Sigmon et al. (2002) found that SAD, S-SAD, and SAD-history participants demonstrated more frequent SCRs and greater SCR magnitude to the winter video relative to participants with nonseasonal depression (MDD) and nondepressed controls.

The aforementioned differences in the stimuli used could help to explain the discrepancy between this study and prior studies (i.e., various light intensities within slides, or winter and summer video scenes). It is possible that participants may have emitted increased general sympathetic arousal in the prior studies based on the content of

the stimuli as opposed to the varying light intensities because the same stimulus scenes were not used in each light condition. In contrast, this study captured the same stimulus scene (e.g., the same tree in the same park, photographed from the same angle) under all light- and season combinations. A second study (Rohan et al., 2003) found no differences between women with SAD history and controls, using the same stimuli as used in the Rohan et al. (2004) study. We do not know whether the Sigmon et al. (2002) study may have confounded seasonal cues with light availability in their video stimuli. In addition, the Maine video was not piloted prior to inclusion in the study in order to establish its content validity. A videotape is arguably a more dynamic stimulus with sounds and movement. It is difficult to distinguish the source or sources of the increased physiological arousal between seasonal cues, possible light intensity, the subject of the scene itself, and auditory stimuli. The length of video stimulus was different from the slide presentation interval used in the present study. The Sigmon et al. (2002) video was continuous and lasted for a period of 10 minutes. Perhaps the allotted time for each block of slides was not sufficient to elicit heightened SCR and SCR magnitude response in a systematic way across the groups. The present study was also unique in simultaneously examining light and seasonal cues as opposed to isolating one factor as these other studies did.

Although it was hypothesized that there would be an increase in zygomatic activity (i.e., smile response) to the bright light and summer slides, zygomatic activity was not consistently different across the groups. The zygomaticus major muscle has been shown to be a marker of reactivity to positive or happy imagery (e.g., Cacioppo et al., 1992; Schwartz et al., 1976a). The lower zygomatic showed no group differences on any

dependent measure (i.e., mean or peak EMG during either stimulus onset or offset). Additionally, the upper zygomaticus showed no group differences on mean EMG response during slide presentation or slide offset. However, there was a surprising finding on the upper zygomatic muscle during slide presentation on peak EMG activity whereby the SAD group responded with greater upper zygomatic activity (i.e., smile response) to bright light/fall slides than controls. Further, a main effect of light within fall for SAD participants was demonstrated whereby SAD emitted greater upper zygomatic activity to bright light/fall stimuli as compared to low light/fall stimuli.

In this case, given that SAD participants appeared to react to bright light/fall stimuli in isolation of any other season, perhaps the significant results represent a statistical anomaly in the data. One explanation for this unexpected finding may be the high degree of variability both between-subjects and within-subjects on peak EMG zygomatic activity during slide presentation (e.g., SAD - bright light/fall, $M = 24.99$, $SD = 25.19$; control – bright light/fall, $M = 9.64$, $SD = 6.57$). These results suggest that peak EMG response may not be the optimal measure of psychophysiological response to affective stimuli. Perhaps future studies may benefit from simultaneous videotaping of participants during the psychophysiological task in order to identify and account for various unpredicted events (e.g., yawn, sneeze, etc.) which could serve to reduce between- and within-subjects variance.

Individual preferences represent another aspect of the present work that may have affected the psychophysiological outcomes. Previous attitude research has examined how individuals form evaluative judgments, based on the positive and negative features of a stimulus (Skowronski & Carlston, 1989; Tesser & Martin, 1996). In an attempt to

extend the processing facilitation concept, Winkielman and Cacioppo (2001) hypothesized that the ease of stimulus processing is directly associated with positive affect. As such, they assessed mood-specific reactivity with facial EMG on the regions associated with happy or pleasant reactions (i.e., zygomatic) and with unhappy or unpleasant reactions (i.e., corrugator) during exposure to black and white line drawings of objects considered to be neutral (e.g., horse, dog, house). Results demonstrated that stimuli that were considered easy-to-process were associated with increased zygomatic activity. There are two suggested reasons why this might be so. First, stimuli that are easier to process may elicit more positive reactions because they accelerate recognition and the potential for cognitive organization (Carver & Scheier, 1990; Vallacher & Nowak, 1999). Second, individuals may feel as if they possess the appropriate knowledge structures to effectively incorporate and cognitively deal with the situation presented (Bless & Fiedler, 1995; Schwarz, 1990).

An application of this construct to the present study concerns the overall ease-of-processing of the included stimuli. Rohan et al. (2003, 2004) and Sigmon et al. (2002) incorporated unidimensional stimuli in their study designs, either light- or season-relevant. Perhaps in the context of this study, the combination of light and season together increased the complexity of the stimulus to a greater degree, whereby it became more difficult for participants to selectively attend to either single dimension of the stimulus scene. This may help to explain the nonsignificant seasonal results on corrugator activity whereby SAD participants only evidenced increased reactivity to low light slides as compared to bright light slides. Each of the slides varies in light intensity and may have resulted in greater attentional focus on the light aspect, as compared to the

season aspect. As was mentioned earlier, the seasonal differences in the stimuli may have been more subtle than the light/dark differences within the slides. If true, and the SAD participants were focused solely on the light dimension of the slides, it seems reasonable to conclude that there would be no significant differences across season on corrugator activity in SAD based on the multidimensional complexity of the slides (i.e., light and season).

There are limitations to this study that should be considered. Given the small sample size, conclusions drawn from these results should be made with caution. In addition, the effect sizes concerning the zygomatic muscle were small, ranging from $\eta^2 = .004$ to $\eta^2 = .078$, suggesting that any differences that might be found with an increased sample size may be too small to be of theoretical or clinical significance.

In general, both within- and between-groups variability was large, as is the nature of psychophysiological data (Cacioppo et al., 2000). Overall, there appeared to be greater within-group variability in the SAD sample than between-groups variability when comparing the SAD and nonseasonal, nondepressed control groups. This increased within-groups variance may be based on individual response stereotopy (Engel, 1960; Lacey & Lacey, 1958). Response stereotopy was evident in that some participants reacted in a peculiar way that was counter to what was hypothesized or expected for a specific contextual stimulus (e.g., increased SCR frequency to light slides in SAD participants). With a larger sample size, not only would power be increased, but there would be enough individuals in each group to identify and control for the variance accounted for by these idiosyncratic responses. Although change scores were considered to help reduce within-groups variability, they were not used when calculating SCR and

EMG because the intent was to measure acute psychophysiological responses on EMG and SCR across blocks of slides. In addition, there were no significant differences between groups at baseline on any of the EMG or skin conductance measures.

Habituation, an important concept for consideration in psychophysiological research, did not appear to negatively affect the present study. In fact, the opposite trend seemed apparent when examining EMG results. For example, at post-hoc inspection of the data, EMG data revealed stronger group differences at later blocks of slides. Results indicated a significant main effect for time from the first block of slides to the last block of slides. Therefore, individuals did not appear to respond initially with heightened EMG response to light- and season-relevant stimuli with later dissipation of response. In an attempt to reduce the likelihood of habituation, the order for the blocks of slides was counterbalanced across participants.

Because of intentional sampling restrictions (i.e., no comorbid Axis I disorders, no current treatment for SAD participants) basic to the execution of a controlled clinical trial in the preliminary treatment development phase, the findings may not be readily generalizable to clinical SAD populations that are more representative of the "real world." In addition, a substantial majority of participants in the present study are women (i.e., 91.7%); therefore, male participants were matched across groups. However, because there is no empirical evidence to date indicating gender differences in EMG reactivity in SAD samples, it is unknown whether results of the present study will be generalizable to male samples.

It is difficult to record surface potentials without possible interference from proximal muscle groups (i.e., there are 37 symmetrical bilaterally-paired muscles in the

cranio-facial area) and the relatively simplistic action of the muscle as a functional unit (i.e., onset of contraction, offset of contraction, and relaxation; Cacioppo et al., 2000); thus, our conclusions about specific muscles (i.e., corrugator or zygomatic) are based on precise measurement and placement of electrodes across participants. A further challenge created by the highly intricate human muscle architecture is the intrusive nature (e.g., restriction of movement, evoking feelings of self-consciousness) of surface EMG recording, which can potentially elicit reactivity, even though the procedure is noninvasive. Despite our best attempts to provide clear and thorough information concerning psychophysiological procedures, coupled with genuine rapport building strategies, some participants verbalized feelings of anticipation or uncertainty concerning the task.

Another limitation in this research effort is the incorporation of a cross-sectional design. This type of study will only provide information concerning whether EMG responses to light and season are correlated with a SAD episode, and in what direction, as opposed to delineating whether the reactivity is a precipitant or consequence of SAD. Similarly, this cross-sectional design will not provide information about causality. For example, differences were found in the SAD group on EMG corrugator reactivity where there was heightened response to low light slides, as compared to bright light slides. However, it is yet unclear whether these findings are due to learning through classical conditioning or perhaps based on well-established dynamic negative core schemas about low light stimuli. Despite this limitation, conducting a cross-sectional study is the first logical step to determine if more sophisticated studies (i.e., longitudinal) are warranted.

There are also other factors that may create laboratory artifacts in EMG studies. First, there are experimenter demand characteristics that could introduce experimental bias among participants. If participants are aware of the hypotheses or expectations of the experimenter, they may consciously or unconsciously change their facial expressions to please the experimenter. A second laboratory artifact may be the presence of evaluation apprehension among participants, which may serve to heighten responsiveness or muscular tension levels. Participants may be somewhat nervous or self-conscious about having numerous electrodes placed on different areas of their bodies (e.g., facial electrodes placed over the corrugator and zygomatic muscles to measure EMG, electrodes placed on medial phalanges to measure SCR and SCR magnitude), a procedure that was novel to most of our participants. However, despite the potential for differences in evaluation among participants, analyses revealed no group differences in baseline EMG or SCR levels. In an attempt to control for these laboratory artifacts, participants were allowed 2-minutes for adaptation where they became used to the recording equipment. In addition, instructions to the participants were explicit, and the experimenters were acutely aware of the need for rapport-building and professionalism, given the nature of the personal contact with participants.

One aspect of human responding to emotional stimuli that has the potential to affect psychophysiological outcomes are individual differences (e.g., age, gender, and expressiveness; Allport & Vernon, 1933). For example, because electrodermal responding is negatively correlated with age, older women who experience negative emotional reactions may not evidence increased skin conductance reactivity because skin conductance measures may not be sensitive enough to detect these changes (Anderson &

McNeilly, 1991). However, in the present study, participants were age, gender, race, and education-matched in order to reduce the impact of these individual differences. In addition, the Vividness of Visual Imagery Questionnaire (VVIQ) was given to each participant after completing the psychophysiological task to assess self-reported imagery ability and there were no significant differences between the groups. There were also no significant differences between females in the groups on current menstrual cycle phase. In addition, no participants in the study reported use of psychotropic medications; therefore, no data was excluded for this reason.

A possible limitation of this psychophysiological study is that participants were not asked explicitly if they were color blind. If any of the included participants were color blind, this may represent a confound for the study. For example, a red/green color blind participant may not have the same response to the fall slides as a participant who could discern the colors of the fall foliage easily. It is possible that a SAD participant who was red/green color blind may have a response pattern to fall stimuli that is similar to that of summer, thereby, contributing to nonsignificant differences between the groups. However, in the present study, it appears as if the light/dark aspect of the stimuli is more salient to participants in general, and color blind participants' nondifferential response to fall stimuli is unlikely to negatively affect the present results.

Another individual difference that has the potential to directly affect the psychophysiological results in this study is current menstrual cycle phase for the female participants. It is well-established that fluctuations in mood, energy, and physiological sensations can vary across the phases of the menstrual cycle, most specifically during the premenstrual, or late phase luteal, phase of the cycle (Altman et al., 1965; Gallant et al.,

1991; Logue & Moos, 1986). This is important given that the present study seeks to measure emotion-specific reactivity. As such, the groups were compared, and there were no significant differences between the proportion of SAD participants (i.e., 2 or 8.0%) and control participants (i.e., 2 or 8.0%) who were in the premenstrual phase of their menstrual cycle while completing the psychophysiological task.

Similarly, Schwartz et al. (1980) demonstrated that females emit exacerbated EMG magnitude as compared to males, which may introduce gender as an artifact. Regarding expressiveness, as previously reviewed, females may be more facially-expressive than males, resulting in greater facial EMG response to negative mood states (Schwartz et al., 1980), thereby increasing individual differences both between and within-groups. However, in the present study, age and gender were matched in the control group and the SAD group.

One further aspect that could influence results is the chronobiologic differences among participants (Greden et al., 1986). Although most individuals' circadian or biological rhythms, are driven by an "intrinsic clock" within a periodicity of 24 hours in length (Aschoff, 1984) phase-shifted (either advanced or delayed) circadian rhythms may affect EMG response. The potential variance introduced based on individual chronobiologic differences was controlled through approximately equal distribution of participants across assessment blocks of time throughout the day (Greden et al., 1986). In this case, participants were assessed between the hours of 7:00 am – 10:00 am, 12:00 pm – 3:00 pm, or 5:00 pm – 8:00 pm. In the context of the present study, because the groups were not perfectly matched across time-of-day, it is possible that some of the

variance in psychophysiological response could be based on individual circadian differences.

Another issue that may represent a weakness of the present study is that the design only incorporates one specific "type" of stimuli, environmental slides, which are visual. Research has shown that various stimuli modalities (e.g., visual, tactile) may be used to elicit differing psychophysiological responses (Cacioppo et al., 2000). Previous results in the SAD literature have shown that visual stimuli may be the "best" stimuli for discriminating light and season and their respective "emotionality" in SAD (Rohan et al., 2004; Sigmon et al., 2002).

Finally, psychophysiological reactivity was compared only across individuals with diagnosed clinical SAD and nonseasonal, nondepressed control participants. A more sophisticated research design would incorporate two control groups: a nonseasonal Major Depression (MDD) group and nonseasonal, nondepressed controls. As outlined in the spectrum of disease model (Lam et al., 2001) the expression of depression in a seasonal or nonseasonal pattern is dependent upon whether an individual has "primary loading" on the depression factor, the seasonality factor, or an intermediate loading on both factors (Lam et al., 2001). Thus, future studies would benefit from including a nonseasonal MDD group that presumably has the vulnerability to depression, but not the vulnerability to seasonality, and a never-depressed control group that has neither a vulnerability to seasonality nor to depression.

Given the overall results of the present research effort, it seems clear that light intensity, as opposed to seasonality, appears to be the more important aspect of the stimuli for this research paradigm in SAD. The visual representations of seasonal stimuli

(i.e., winter) were significant when assessing POMS Depression-Dejection subscale scores; however, they were not significant using either SCR or EMG as measures of general and emotion-specific psychophysiological arousal. Given the saliency and greater degree of personal relevancy of the light-relevant component of the present stimuli, future SAD studies would benefit from inclusion of stimuli with varying light intensities as the primary focus.

Future studies may benefit from assessment of reactivity to light cues across the seasons (i.e., spring/summer versus fall/winter) to examine any differences between reactivity in an episodic and a remitted period. However, previous SAD researchers found no significant differences in psychophysiological reactivity in an S-SAD sample, regardless of the season (Rohan et al., 2003). Participants' reactivity appeared to be in response to low light cues, as opposed to bright light or ambiguous light cues. Another study found no differences in SCR magnitude in SAD or nondepressed controls across the seasons (i.e., summer, fall, winter;
Rohan et al., 2004). Consequently, previous findings provide evidence that heightened psychophysiological response to light-relevant stimuli in SAD may be classically-conditioned and, therefore, may represent a sensitivity to light-relevant cues, regardless of the season in which the assessment is completed.

One approach for future psychophysiological research that may improve upon the present study design is to include a neutral slide series, varying in light intensity only. For example, adding a block of slides with neutral content (e.g., abstract drawings) in both low light and bright light conditions may provide additional information concerning the greater saliency of the light aspect of the stimuli. This is important, especially given

that the present results are nonsignificant with regards to differences in psychophysiological responding to seasonal stimuli.

In addition, the difference in psychophysiological responding between slide presentation (i.e., 10 seconds) and slide offset (i.e., 10 seconds) may reflect position of the block of slides within the overall task, after-effects of a block of slides, or anticipatory contraction. Future studies may benefit from including a 2 - 3 minute baseline period (where no slides are presented) after each block of slides. In the present study, there was evidence of a psychophysiological "baseline shift" or increase in reactivity from earlier to later blocks of slides. After presentation of blocks of slides, EMG and SCR decreased after each block of slides, but never returned to a level reflective of natural baseline responding. If future study designs incorporate a baseline period after presentation of each block of slides, the effects of baseline shift will be minimized and more accurate change scores can be computed, perhaps giving a more accurate overall comparison of psychophysiological responding across the various slide-types.

Other future research studies should consider incorporation of a new technique called automated facial imaging (Cohn & Kanade, in press). This technique uses "computer vision," a method of coding facial muscle activity that simultaneously recognizes perceptually meaningful patterns. The intent is to classify emotion-specific expressions, and more importantly, to capture facial Action Units (AU), the smallest changes in facial expression that can be visibly discriminated. Use of this method in future studies of facial expression analyses, may prove to be a more sensitive measure than facial EMG, because electrodes placed on individuals' faces may result in inhibitory

responses. This technique may also be useful in studies of behavioral assessment and emotion (Cohn & Kanade, in press).

Another area to explore in future studies is that of "Duchenne's smile," which encompasses activity within the orbicularis oculi (located directly under the eye) in conjunction with the zygomaticus major. Research studies demonstrating simultaneous activity in these two muscle groups have been linked to smiles associated with enjoyment in different settings and on diverse measures (Frank, Ekman, & Friesen, 1993). Specifically, when participants produced an "enjoyment smile," which incorporates the Duchenne marker, they reported that they "felt" more enjoyment when exposed to happy or pleasant stimuli, as compared to smiles that did not reveal activity in Duchenne's marker (i.e., a non-enjoyment smile; Eckman, Davidson, & Friesen, 1990). In addition, Eckman et al. (1990) reported that it is the degree of zygomatic activity in combination with orbicularis activity that predicts participants self-reported ratings of happiness, rather than overall zygomaticus reactivity alone. Tests of this construct may be more sensitive in detecting positive affective emotion to environmental stimuli in SAD

The main results of this study suggest that there may be differential EMG response patterns to affect-laden stimuli in individuals with SAD. Specifically, the present findings consistently revealed differences between SAD and control participants in their reaction to light and dark stimuli across the various outcome measures (i.e., EMG corrugator, POMS, and perceived pleasantness ratings), suggesting that low light stimuli may intensify depressive affect and bright light stimuli may have a mood-enhancing effect on individuals with SAD. Overall, results suggest that light intensity may be a more salient cue than season in determining transient mood-shifts in SAD when

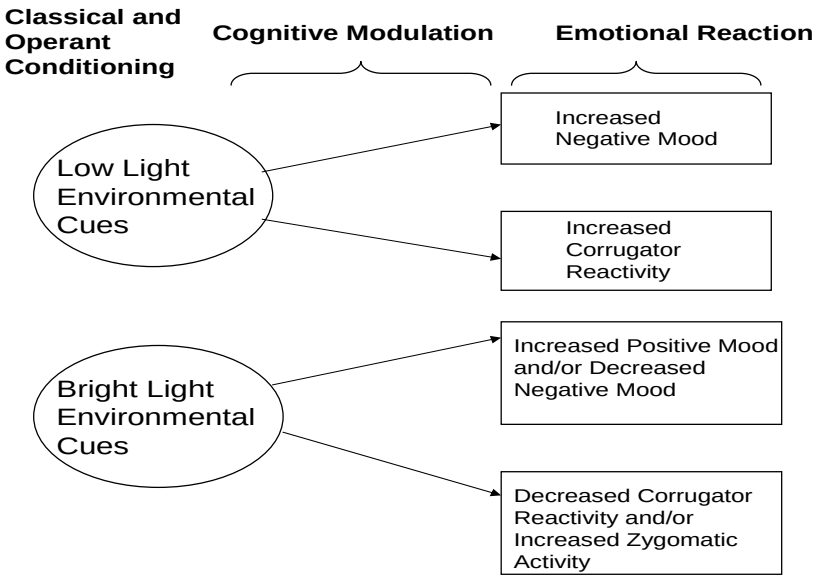
appraising the environment. Consistent with Rohan's (2002) integrative cognitive-behavioral model, it appears as if individuals with SAD may indeed experience increased psychophysiological arousal and depressive affect when exposed to environmental stimuli of low light intensity such as those repeatedly paired with the onset of a SAD episode. Consequently, it seems evident that surface facial EMG (i.e., corrugator) is particularly sensitive and well-suited to capturing negative affective reactivity in a SAD sample in response to low light stimuli and should be explored in future psychophysiological studies.

As a framework for understanding the present results and as a model for future studies, a conceptual model is proposed that integrates cognitive modulation, classical, and operant conditioning (See Figure 7). Borrowing from Rohan's (2002) integrative cognitive-behavioral model, it is assumed that classical conditioning has occurred, whereby reduced light availability has been repeatedly paired with the onset of a depressive episode among individuals with SAD. Through the process of generalization, environmental cues depicting low light conditions become conditioned stimuli for depressive behavior. This is why showing a SAD-vulnerable individual a picture of a dark, dreary sky can produce a negative shift in mood and affect. Similarly, increased photoperiod and light availability have been repeatedly paired with amelioration of depressed mood in SAD. Environmental cues related to sunshine may trigger a positive mood shift. Through stimulus generalization, this can even occur in reaction to a photograph of a sunny, clear sky. Operant conditioning may also be at play. If bright, sunny environments are associated with positive reinforcement for SAD individuals, it

follows that they would show approach behavior towards sunshine (i.e., try to place themselves in sunny settings to maximize positive reinforcement).

***Figure 7.***

**Integrative Model of Emotional and Psychophysiological Reactivity to Light-Relevant Stimuli in SAD.**




In contrast, if dark environments are related to negative emotions, individuals with SAD may show avoidance and/or escape when faced with low light environmental cues to reduce their distress via negative reinforcement. This is consistent with anecdotal observations of "hibernation-like" behavior in SAD. Beck (1967, 1976) theorized that negative beliefs are learned. Therefore, through classical and operant conditioning, negative core beliefs about low light availability and positive core beliefs about bright light may develop in SAD. These core beliefs are likely to be dichotomous (i.e., light is good, dark is bad, Rohan et al., 2003). Core beliefs may become a cognitive mediator between exposure to a light-relevant stimulus and emotional response. Once in place, it

should take progressively less and less saliency of a stimulus to activate this process (e.g., more subtle light-relevant cues can be cognitively processed and lead to an emotional state).

# TABLES

***Table 1.***

Studies That Link Surface Facial EMG Responses to Specific Emotions.

| **Authors** | **Participants (*n*)** | **Stimuli** | **Results** |
|---|---|---|---|
| Cacioppo et al. (1992) | Nondepressed undergraduate Women (20) | Pleasant/unpleasant/neutral facial expressions and scenery<br>* Amplify-expression instructions<br>* Inhibit-expression instructions<br>* No verbal instructions | 1) Corrugator activity lower $F$ (1.2, 19.2) = 19.03 and zygomatic activity higher $F$ (1.2, 18.5) = 19.82 when viewing pleasant stimuli (faces or scenes), $p$s < .001<br>2) Corrugator and zygomatic EMG activity highest in "amplify" condition<br>*↓in corrugator region and ↑in zygomatic region when viewing pleasant stimuli<br>3) Corrugator and zygomatic EMG activity lowest in "inhibit" condition<br>*↓in corrugator when participants viewed pleasant stimuli as compared to neutral or unpleasant stimuli<br>4) EMG zygomatic activity did not differentiate positive from negative stimuli<br>5) Facial EMG did not differ in response to slides of nature versus social scenes matched for pleasantness |
| Greden et al. (1986) | Patients w/endogenous depression (37)<br>Patients w/non-endogenous depression (26)<br>Female nondepressed controls (29) | Happy/sad personally-relevant imagery and "typical day" imagery | 1) Normal control corrugator – significant differences from baseline to typical day ($p$ < .05) and baseline to sad (p < .001); normal control zygomatic – significant differences from baseline to happy ($p$ < .001), baseline to typical day ($p$ <.05) and baseline to sad ($p$ <.01)<br>2) Endogenous depressed corrugator – significant differences from baseline to happy (p < .05) and baseline to sad (p < .001); endogenous depressed zygomatic – no significant differences between baseline and any imagery condition |

| Authors | Type/No. Participants | Stimuli | Results |
|---|---|---|---|
| | | | 3) Nonendogenous depressed corrugator – no significant differences between baseline and any imagery condition; nonendogenous depressed zygomatic – significant differences between baseline and typical day condition ($p < .01$)<br>4) When compared with normal participants, both endogenous and nonendogenous participants showed a relative lack of activity in both corrugator and zygomatic regions to different stimuli<br>5) Significant differences between endogenous and nonendogenous depressed patients for corrugator happy ($p < .05$) and corrugator sad ($p < .05$) |
| Oliveau & Willmuth (1979) | Inpatient Depressed psychiatric inpatients<br>Nondepressed psychiatric control participants<br>21 women; 19 men | Recall and "picture in your mind" sad/happy/typical day events experienced in your life | 1) Significant ↑corrugator activity from baseline in the total group (depressed and nondepressed) when asked to "picture in their mind" a sad circumstance<br>2) No change from baseline corrugator activity when asked to imagine happy circumstances, regardless of group<br>3) Both depressed and nondepressed participants grouped according to Zung Self-Rating Depression Scale (SDS) evidenced significant ↑ EMG corrugator activity from baseline in typical day imagery condition<br>4) Unable to distinguish between sad and typical day imagery states |
| Schwartz et al. (1976a) | Depressed (12)<br>Nondepressed controls (12) | Happy/sad personally-relevant imagery<br>* "Think" about imagery<br>* "Reexperience the feelings" | 1) Happy imagery condition - corrugator activity ↓ below baseline and zygomatic activity ↑ from baseline in total sample<br>2) Sad imagery condition – corrugator activity ↑ above baseline and little effect on zygomatic activity in total sample<br>3) Differences between happy versus sad for both corrugator ($p < .01$) and zygomatic ($p < .01$) in "think" condition |

| Authors | Type/No. Participants | Stimuli | Results |
|---|---|---|---|
| | | | 4) Depressed participants produce reliable ↑ EMG zygomatic in the "feel" happy condition ($p < .01$)<br>5) When comparing happy versus sad separately for "think" and "feel" conditions, depressed participants evidence significant corrugator activity ($p < .08$)<br>6) In the "feel" condition, depressed participants generate reliable differences in corrugator ($p < .01$) and zygomatic ($p < .05$) muscle activity |
| Schwartz et al. (1976b) | Depressed females (12)<br>Nondepressed females (12) | Happy/sad personally-relevant imagery and "typical day" imagery | 1) Happiness associated w/↓ corrugator activity from baseline in total sample ($p < .05$)<br>2) Sadness associated w/↑ corrugator activity from baseline in total sample ($p < .01$)<br>3) Corrugator activity differentiates happiness from both sadness ($p < .01$) and anger ($p < .01$) in depressed and nondepressed participants<br>4) Depressed participants - ↓corrugator activity in "happy" condition relative to nondepressed controls<br>5) In "typical day" condition, depressed evidenced "sad" pattern; nondepressed exhibited "happy" pattern<br>6) Self-evaluation of mood states: Nondepressed reported ↑ happiness in happy imagery ($p < .002$) and less sadness during typical day ($p < .002$) as compared to depressed participants |

| Authors | Type/No. Participants | Stimuli | Results |
|---|---|---|---|
| Sloan et al. (2002) | Dysphoric undergraduate Students<br>Nondysphoric undergraduate Students (42 females) | Happy and unhappy facial expressions from the IAPS (Lang et al., 1999) | 1) Dysphoric students evidenced significantly ↓ zygomatic activity when viewing happy facial expressions compared to nondysphoric students ($p < .05$)<br>2) No significant differences were found when comparing dysphoric and nondysphoric students on zygomatic reactivity to unhappy facial expressions<br>3) Dysphoric students evidenced significantly ↑ corrugator activity when viewing the happy facial expressions during the first 2 seconds of viewing as compared to the nondysphoric students ($p < .05$)<br>4) Both dysphoric and nondysphoric students evidenced ↑ corrugator EMG reactivity when viewing unhappy facial expressions |

***Table 2.***

**POMS Depression-Dejection Subscale Change Scores From Baseline to Stimuli Offset – Pilot Study 2.**

| Season (Light Intensity) | POMS Δ Scores *M* (*SD*) | Median Δ Scores |
|---|---|---|
| Summer | | |
| Bright Light | 5.87 (6.45) | 5.0* |
| Low Light | .80 (5.97) | 1.0 |
| Fall | | |
| Bright Light | 3.27 (4.01) | 3.0 |
| Low Light | .47 (4.87) | 0.0 |
| Winter | | |
| Bright Light | 3.20 (7.76) | 3.0 |
| Low Light | -2.40 (7.06) | -3.0* |

*$p < .01$

***Table 3.***

**Pleasantness Rating Scores to Stimuli Offset – Pilot Study 2.**

| Season (Light Intensity) | Pleasantness Ratings *M (SD)* |
|---|---|
| Summer | |
| Bright Light | 5.17 (1.47)* |
| Low Light | 3.58 (1.56) |
| Fall | |
| Bright Light | 4.67 (.78) |
| Low Light | 3.25 (.87) |
| Winter | |
| Bright Light | 4.25 (1.36) |
| Low Light | 2.42 (1.08)* |

*$p < .001$

***Table 4.***

**Participant Demographics.**

| | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Age, *M (SD)* | 41.58 (11.7) | 41.83 (12.75) |
| Gender, No. (%) | | |
| Female | 22 (91.7) | 22 (91.7) |
| Male | 2 (8.3) | 2 (8.3) |
| Race, No. (%) | | |
| Asian | 2 (8.3) | 0 (0.0) |
| African American | 4 (16.7) | 4 (16.7) |
| Hispanic | 0 (0.0) | 2 (8.3) |
| Caucasian | 17 (70.8) | 17 (70.8) |
| Other | 1 (4.2) | 1 (4.2) |
| Marital Status, No. (%) | | |
| Single | 8 (33.3) | 7 (29.2) |
| Married | 11 (45.8) | 16 (66.7) |
| Living Together | 4 (16.7) | 0 (0.0) |
| Divorced | 1 (4.2) | 1 (4.2) |
| Education, No. (%) | | |
| Graduated H.S. | 0 (0.0) | 2 (8.3) |
| Some College | 6 (25.0) | 4 (16.7) |
| Graduated College | 8 (33.3) | 6 (25.0) |

| | | |
|---|---|---|
| Some Graduate School | 3 (12.5) | 6 (25.0) |
| Completed Graduate School | 7 (29.2) | 6 (25.0) |
| Employment, No. (%) | | |
| Retired | 0 (0.0) | 1 (4.2) |
| Homemaker | 1 (4.2) | 1 (4.2) |
| Business | 7 (29.2) | 1 (4.2) |
| Professional | 3 (12.5) | 5 (20.8) |
| Other Medical Professional | 0 (0.0) | 1 (4.2) |
| Other | 13 (54.2) | 15 (62.5) |

***Table 5.***

**Mean EMG Corrugator Supercilii Activity, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 7.85 (.78) | 9.88 (.87) |
| Low Light | 10.41 (1.25) | 10.11 (.79) |
| Fall | | |
| Bright Light | 9.09 (1.01) | 11.02 (1.11) |
| Low Light | 10.34 (1.22) | 11.18 (.79) |
| Winter | | |
| Bright Light | 10.42 (1.25) | 11.30 (.84) |
| Low light | 11.26 (1.23) | 11.03 (.93) |
| Collapsed Across Seasons | | |
| Bright Light | 9.12 (1.01)* | 10.73 (.94) |
| Low Light | 10.67 (1.23)* | 10.77 (.84) |

* Significant light main effect, $F$ (1, 46) = 14.20, $p < .001$, $\eta^2 = .24$

***Table 6.***

**Mean EMG Corrugator Supercilii Activity, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 7.92 (.76) | 9.90 (.84) |
| Low Light | 10.12 (1.19) | 10.41 (.83) |
| Fall | | |
| Bright Light | 9.05 (1.07) | 10.99 (1.03) |
| Low Light | 10.30 (1.24) | 10.79 (.73) |
| Winter | | |
| Bright Light | 10.08 (1.21) | 11.18 (.80) |
| Low light | 11.21 (1.30) | 10.95 (.88) |
| Collapsed Across Seasons | | |
| Bright Light | 9.02 (1.01)* | 10.69 (.89) |
| Low Light | 10.54 (1.24)* | 10.72 (.81) |

* Significant light main effect, $F$ (1, 46) = 13.75, $p$ = .001, $\eta^2$ = .23

***Table 7.***

**Peak EMG Corrugator Supercilii Activity, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 19.38 (1.92) | 24.21 (1.84) |
| Low Light | 25.81 (2.39) | 28.00 (3.15) |
| Fall | | |
| Bright Light | 24.71 (2.27) | 28.90 (3.29) |
| Low Light | 26.09 (2.93) | 25.77 (2.50) |
| Winter | | |
| Bright Light | 25.43 (2.91) | 23.07 (1.85) |
| Low light | 25.32 (2.40) | 27.51 (1.94) |

***Table 8.***

**Peak EMG Corrugator Supercilii Activity, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 18.85 (1.97) | 23.95 (1.95) |
| Low Light | 23.59 (2.55) | 27.80 (3.27) |
| Fall | | |
| Bright Light | 20.34 (2.08) | 25.05 (2.09) |
| Low Light | 23.01 (2.60) | 27.63 (2.76) |
| Winter | | |
| Bright Light | 20.86 (2.08) | 25.08 (2.08) |
| Low light | 24.62 (1.93) | 24.02 (2.16) |

***Table 9*.**

**Mean EMG Lower Zygomaticus Major Activity, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 5.97 (1.06) | 3.75 (.53) |
| Low Light | 4.64 (.67) | 3.38 (.46) |
| Fall | | |
| Bright Light | 5.95 (1.26) | 3.07 (.25) |
| Low Light | 3.82 (.67) | 3.54 (.68) |
| Winter | | |
| Bright Light | 6.30 (1.32) | 3.59 (.51) |
| Low light | 4.53 (.81) | 2.96 (.37) |

***Table 10*.**

**Mean EMG Lower Zygomaticus Major Activity, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 4.99 (.90) | 3.80 (.48) |
| Low Light | 4.12 (.76) | 3.22 (.43) |
| Fall | | |
| Bright Light | 4.63 (.70) | 3.23 (.38) |
| Low Light | 3.72 (.67) | 3.19 (.44) |
| Winter | | |
| Bright Light | 5.17 (.97) | 3.32 (.44) |
| Low light | 4.27 (.80) | 2.86 (.27) |

***Table 11.***

**Peak EMG Lower Zygomaticus Major Activity, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 19.56 (3.90) | 14.58 (3.01) |
| Low Light | 16.68 (2.80) | 14.03 (3.70) |
| Fall | | |
| Bright Light | 22.79 (5.36) | 11.65 (2.25) |
| Low Light | 12.78 (2.46) | 13.74 (3.59) |
| Winter | | |
| Bright Light | 24.02 (5.71) | 17.19 (2.92) |
| Low light | 13.32 (2.18) | 12.17 (2.69) |

***Table 12*.**

**Peak EMG Lower Zygomaticus Major Activity, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 14.30 (1.75) | 16.32 (3.03) |
| Low Light | 14.55 (2.99) | 13.22 (2.66) |
| Fall | | |
| Bright Light | 14.67 (1.87) | 12.74 (2.76) |
| Low Light | 12.57 (2.38) | 11.59 (2.05) |
| Winter | | |
| Bright Light | 15.27 (2.48) | 16.07 (3.18) |
| Low light | 13.69 (2.33) | 12.76 (2.73) |

***Table 13.***

**Mean EMG Upper Zygomaticus Major Activity, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 5.07 (.67) | 3.89 (.67) |
| Low Light | 4.75 (.63) | 3.66 (.70) |
| Fall | | |
| Bright Light | 5.58 (.91) | 3.41 (.49) |
| Low Light | 3.72 (.64) | 3.49 (.64) |
| Winter | | |
| Bright Light | 8.34 (2.24) | 3.32 (.39) |
| Low light | 6.34 (1.90) | 3.09 (.39) |

***Table 14.***

**Mean EMG Upper Zygomaticus Major Activity, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 4.08 (.45) | 4.22 (.73) |
| Low Light | 4.18 (.57) | 3.68 (.51) |
| Fall | | |
| Bright Light | 4.55 (.53) | 3.96 (.70) |
| Low Light | 3.64 (.57) | 3.48 (.54) |
| Winter | | |
| Bright Light | 6.97 (1.90) | 3.56 (.52) |
| Low light | 5.86 (1.80) | 3.25 (.40) |

***Table 15*.**

**Peak EMG Upper Zygomaticus Major Activity, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 17.73 (3.49) | 18.91 (4.66) |
| Low Light | 20.13 (4.84) | 17.81 (5.57) |
| Fall | | |
| Bright Light | 24.99 (5.14) [a, b] | 9.64 (1.34) [a] |
| Low Light | 16.55 (4.53) [b] | 13.63 (3.69) |
| Winter | | |
| Bright Light | 26.09 (5.09) | 14.41 (3.10) |
| Low light | 18.80 (3.95) | 12.83 (2.99) |

[a] Significant group main effect, $F$ (1, 46) = 8.34, $p$ = .006, $\eta^2$ = .15

[b] Significant light main effect, $F$ (1, 46) = 8.60, $p$ = .005, $\eta^2$ = .16

***Table 16.***

**Peak EMG Upper Zygomaticus Major Activity, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 13.04 (1.62) | 18.35 (3.76) |
| Low Light | 16.50 (4.57) | 17.33 (4.14) |
| Fall | | |
| Bright Light | 18.57 (3.30) | 16.18 (3.92) |
| Low Light | 20.04 (5.57) | 14.76 (3.37) |
| Winter | | |
| Bright Light | 18.96 (3.25) | 17.85 (4.66) |
| Low light | 18.48 (3.73) | 16.23 (3.97) |

***Table 17.***

**Significant Skin Conductance Response Frequency, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | .55 (.12) | .33 (.06) |
| Low Light | .39 (.08) | .35 (.09) |
| Fall | | |
| Bright Light | .43 (.10) | .24 (.07) |
| Low Light | .37 (.12) | .30 (.08) |
| Winter | | |
| Bright Light | .47 (.12) | .23 (.07) |
| Low light | .49 (.11) | .50 (.10) |
| Collapsed Across Seasons | | |
| Bright Light | .48 (.11) | .27 (.07)* |
| Low Light | .42 (.10) | .38 (.09)* |

* Significant light main effect, $F$ (1, 46) = 6.99, $p$ = .011, $\eta^2$ = .13

***Table 18*.**

**Significant Skin Conductance Response Frequency, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | .34 (.09) | .33 (.08) |
| Low Light | .29 (.11) | .36 (.09) |
| Fall | | |
| Bright Light | .37 (.11) | .26 (.09) |
| Low Light | .27 (.09) | .24 (.06) |
| Winter | | |
| Bright Light | .36 (.10) | .32 (.07) |
| Low light | .39 (.12) | .38 (.12) |

***Table 19*.**

**Significant Skin Conductance Response Magnitude, During Slide Presentation, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | .05 (.02) | .01 (.00) |
| Low Light | .03 (.01) | .03 (.01) |
| Fall | | |
| Bright Light | .03 (.01) | .01 (.01) |
| Low Light | .02 (.01) | .01 (.01) |
| Winter | | |
| Bright Light | .04 (.01) | .01 (.01) |
| Low light | .03 (.01) | .03 (.01) |
| Collapsed Across Seasons | | |
| Bright Light | .04 (.01)* | .01 (.01)* |
| Low Light | .03 (.01) | .02 (.01) |

* Significant group main effect, $F$ (1, 46) = 6.36, $p$ = .015, $\eta^2$ = .12

***Table 20*.**

**Significant Skin Conductance Response Magnitude, During Slide Offset, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | .02 (.01) | .02 (.01) |
| Low Light | .03 (.01) | .02 (.01) |
| Fall | | |
| Bright Light | .02 (.01) | .03 (.01) |
| Low Light | .02 (.01) | .02 (.01) |
| Winter | | |
| Bright Light | .03 (.01) | .03 (.01) |
| Low light | .03 (.01) | .02 (.01) |

***Table 21.***

**POMS Depression-Dejection Subscale Change Scores From Baseline, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 5.58 (1.27) | .17 (.14) |
| Low Light | -4.58 (1.45) | -1.12 (.46) |
| Fall | | |
| Bright Light | 2.75 (1.26) | .13 (.17) |
| Low Light | -4.71 (1.80) | -.71 (.39) |
| Winter | | |
| Bright Light | -.04 (1.22) | -.42 (.33) |
| Low light | -7.50 (1.64) | -1.79 (.63) |
| Collapsed Across Seasons | | |
| Bright Light | 2.76 (1.25) [a, b] | -.04 (.21) [b] |
| Low Light | -5.60 (1.63) [a, c] | -1.21 (.49) [c] |
| Collapsed Across Light | | |
| Summer | .50 (1.36) [d] | -.48 (.30) |
| Fall | -.98 (1.53) [e] | -.29 (.28) |
| Winter | -3.77 (1.43) [d, e, f] | -1.11 (.48) [f] |

[a] Significant light main effect, $F$ (1, 46) = 80.36, $p$ < .001, $\eta^2$ = .64

[b] Significant group main effect, $F$ (1, 46) = 8.92, $p$ = .005, $\eta^2$ = .16

[c] Significant group main effect, $F$ (1, 46) = 8.90, $p$ = .005, $\eta^2$ = .16

[d] Significant season main effect, $F$ (2, 45) = 14.12, $p$ < .001, $\eta^2$ = .39; $t$(23) = -3.83, $p$ = .001

[e] Significant season main effect, $F$ (2, 45) = 14.12, $p$ < .001, $\eta^2$ = .39; $t$(23) = -2.10, $p$ = .047

[f] Significant group main effect, $F$ (1, 46) = 3.88, $p$ = .055, $\eta^2$ = .08

***Table 22*.**

**Pleasantness Rating Scores, M (SEM).**

| Light Intensity | SAD ($n$ = 24) | Control ($n$ = 24) |
|---|---|---|
| Summer | | |
| Bright Light | 6.00 (.18) [c] | 5.79 (.19) [d] |
| Low Light | 2.38 (.25) [a, c] | 3.58 (.25) [a, d] |
| Fall | | |
| Bright Light | 5.21 (.28) [e] | 5.67 (.21) [f] |
| Low Light | 2.88 (.20) [b, e] | 4.59 (.23) [b, f] |
| Winter | | |
| Bright Light | 3.88 (.30) | 4.75 (.29) |
| Low light | 2.21 (.26) | 3.42 (.26) |

[a] Significant group main effect, $F$ (1, 46) = 11.60, $p$ = .001, $\eta^2$ = .20

[b] Significant group main effect, $F$ (1, 46) = 30.71, $p$ < .001, $\eta^2$ = .40

[c] Significant light main effect, $F$ (1, 46) = 117.39, $p$ < .001, $\eta^2$ = .72

[d] Significant light main effect, $F$ (1, 46) = 43.57, $p$ < .001, $\eta^2$ = .49

[e] Significant light main effect, $F$ (1, 46) = 98.27, $p$ < .001, $\eta^2$ = .68

[f] Significant light main effect, $F$ (1, 46) = 21.18, $p$ < .001, $\eta^2$ = .32

***Table 23.***

**Female Participant Menstrual Cycle Phase, During Assessment.**

| | SAD ($n$ = 24) | Control ($n$ =24) |
|---|---|---|
| Cycle Phase, No. (%) | | |
| Premenstrual | 2 (8.3) | 2 (8.3) |
| Menstrual | 5 (20.8) | 1 (4.2) |
| Other Phase | 11 (45.9) | 13 (54.2) |
| No Cycle | 1 (4.2) | 1 (4.2) |
| Menopause | 2 (8.3) | 5 (20.8) |
| Missing | 3 (12.5) | 2 (8.3) |

***Table 24.***

**Time-of-Day For Assessment.**

| | SAD ($n$ = 24) | Control ($n$ =24) |
|---|---|---|
| Assessment Time, No. (%) | | |
| 7:00 – 10:00 am | 7 (29.2) | 5 (20.8) |
| 12:00 – 3:00 pm | 4 (16.7) | 11 (45.8) |
| 5:00 – 8:00 pm | 13 (54.2) | 8 (33.3) |

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## APPENDIX A

### Assessment of Menstrual Cycle Phase (AMCP)

In pre-menopausal women, mood and behavior can change across different phases of the menstrual cycle. Cycle phase may impact some women's responses to some of the measures we are collecting today. Therefore, we ask you to take a few minutes to answer the following questions concerning your menstrual cycle. Please refer to the calendar as needed.

1. Have you reached menopause yet (circle one)? YES NO

2. If not, what was the start date of your last period (the day you actually started bleeding)? _____

2. On average, how many days does your menstrual cycle last? _____

3. Are you current taking any type of birth control (circle one)? YES NO

4. If yes, what type of birth control? _____

## APPENDIX B

## Vividness of Visual Imagery Questionnaire (VVIQ)

Name: _____ Gender (circle one): Male Female

Occupation (If student, then give course of study and stage reached): _____

Visual imagery refers to the ability to visualize, that is, the ability to form mental pictures, or to "see in the mind's eye." Marked individual differences have been found in the strength and clarity of reported visual imagery and these differences are of considerable psychological interest.

The aim of this test is to determine the vividness of your visual imagery. The items of the test will possibly bring certain images to your mind. You are asked to rate the vividness of each image by reference to the 5-point scale given below. For example, if your image is "vague and dim" then give it a rating of 4. After each item, write the appropriate number on the line provided. The line is provided for an image obtained with your eyes open. Before you turn to the questionnaire items, familiarize yourself with the different categories on the rating scale. Throughout the test, refer to the rating scale when judging the vividness of each image. Try to do each item separately, independent of how you may have done other items. Complete all items for images obtained with the eyes open.

### Rating Scale

### The image aroused by an item might be:

| | |
|---|---|
| Perfectly clear and as vivid as normal vision | **Rating 1** |
| Clear and reasonably vivid | **Rating 2** |
| Moderately clear and vivid | **Rating 3** |
| Vague and dim | **Rating 4** |
| No image at all, you only "know" that you are thinking of an object | **Rating 5** |

---

In answering items 1 to 4, think of some relative or friend whom you frequently see (but who is not with you at present) and consider carefully the picture that comes before your mind's eye.

**1** The exact contour of face, head, shoulders, and body. _____
**2** Characteristic poses of head, attitudes of body, etc. _____
**3** The precise carriage, length of step, etc. in walking. _____
**4** The different colors worn in some familiar clothes. _____

**……**

Visualize the rising sun. Consider carefully the picture that comes before your mind's eye.

**5** The sun is rising above the horizon into a hazy sky. _____
**6** The sky clears and surrounds the sun with blueness. _____
**7** Clouds. A storm blows up, with flashes of lightening. _____
**8** A rainbow appears. _____

……..

Think of the front of a shop which you often go to. Consider the picture that comes before your mind's eye.

**9** The overall appearance of the shop from the opposite side of the road. _____
**10** A window display including colors, shape, and details of individual items for sale. _____
**11** You are near the entrance. The color, shape, and details of the door. _____
**12** You enter the shop and go to the counter. The counter assistant serves you. Money changes hands. _____

……

Finally, think of a country scene which involves trees, mountains, and a lake. Consider the picture that comes to your mind's eye.

**13** The contours of the landscape. _____
**14** The color and shape of the trees. _____
**15** The color and shape of the lake. _____
**16** A strong wind blows on the tree and on the lake causing waves. _____

## APPENDIX C

### Profile of Mood States (POMS)

**Directions:** Below is a list of words that describe feelings that people have. Please read each one carefully. Then select the number that best describes HOW YOU FEEL RIGHT NOW. Place that number on the small line to the left of each word. Do not skip any items, and print your numbers clearly.

0 = Not at all
1 = A little
2 = Moderately
3 = Quite a bit
4 = Extremely

_____ 1. Tense
_____ 2. Unhappy
_____ 3. Sorry for things done
_____ 4. Shaky
_____ 5. Sad
_____ 6. On edge
_____ 7. Blue
_____ 8. Panicky
_____ 9. Hopeless
_____ 10. Relaxed
_____ 11. Unworthy
_____ 12. Uneasy
_____ 13. Restless
_____ 14. Discouraged
_____ 15. Nervous
_____ 16. Lonely
_____ 17. Miserable
_____ 18. Anxious
_____ 19. Gloomy
_____ 20. Desperate
_____ 21. Helpless
_____ 22. Worthless
_____ 23. Terrified
_____ 24. Guilty

# APPENDIX D

## Perceived Pleasantness Ratings

Please rate the pictures you just saw on the following scale. (Circle one).

| 1 | 2 | 3 | 4 | 5 | 6 | 7 |
|---|---|---|---|---|---|---|
| very unpleasant | | | | | | very pleasant |

# APPENDIX E

## Slide Randomization Sequence

Sequence 1

1. Bright Light/Summer
2. Low Light/Summer
3. Bright Light/Fall
4. Low Light/Fall
5. Bright Light/Winter
6. Low Light/Winter

Sequence 2

1. Low Light/Summer
2. Bright Light/Summer
3. Low Light/Fall
4. Bright Light/Fall
5. Low Light/Winter
6. Bright Light/Winter

Sequence 3

1. Bright Light/Winter
2. Low Light/Winter
3. Bright Light/Summer
4. Low Light/Summer
5. Bright Light/Fall
6. Low Light/Fall

Sequence 4

1. Low Light/Winter
2. Bright Light/Winter
3. Low Light/Summer
4. Bright Light/Summer
5. Low Light/Fall
6. Bright Light/Fall

Sequence 5

1. Bright Light/Fall
2. Low Light/Fall
3. Bright Light/Winter
4. Low Light/Winter
5. Bright Light/Summer
6. Low Light/Summer

Sequence 6

1. Low Light/Fall
2. Bright Light/Fall
3. Low Light/Winter
4. Bright Light/Winter
5. Low Light/Summer
6. Bright Light/Summer

# APPENDIX F

## Psychophysiological Recording Procedures

### Profile of Mood States

Gather the POMS packet (#0 - #6) and put them on a clipboard with a pen.

### Instructions to Participants

Before you begin attaching any equipment, say the following to the participant:

> *In the next task, we will measure your physical reactions to outdoor photographs. You will view the photographs while seated comfortably, and we will measure your skin conductance (how much your palms sweat), pulse, breathing rate, and how tense your muscles in your face become. These measurements are taken by attaching small disks and straps to two of your fingers, one ankle, one wrist, your chest, your forehead, and your cheeks. This is not painful or harmful in any way. After we connect these disks and straps, you will have about 5 minutes sit and relax to get used to how it feels. After this 5-minute resting period, I will ask you to complete this questionnaire to rate your mood. Then, you will view several photographs on the computer screen of various outdoor scenes. Each photograph will be shown for a few seconds, followed by a blank screen for a few seconds. I would like for you to relax and imagine what it would feel like if you were actually in the picture. Imagine what you would be feeling and thinking if you were really there. After you look at five similar pictures, the computer screen will pause, and I will ask you to rate that group of pictures considering how pleasant or unpleasant they were for you on this 7-point rating scale and to rate your mood again. I will then restart the computer and the process will be repeated five more times, with five different groups of similar pictures. Do you have any questions or concerns before we start?*

### Preparation

First, the skin must be properly prepared for electrode placement. Use the squirt bottle to wet a facial cleansing cloth and give it to the participant to soap their left check, above their left eyebrow, the middle of their forehead, their hands, right wrist, and left ankle. Wet a paper towel for them to wipe off the soap from the cleanser. After the area is dried, the same areas are cleansed with an alcohol prep pad (called “electrode prep pad”).

NOTE: If the participant is a male with significant facial hair (e.g., beard or moustache) that may interfere with facial EMG electrode placement, do not record EMG. Note on

the participants POMS that EMG was not recorded due to facial hair, but DO measure SCR, body temperature, respiration, and EKG.

For EMG only, the skin on the left cheek and above the left eyebrow should then be abraded with NUPREP skin prep gel paste swirled on the skin for 20 to 30 seconds with a Q-tip (the skin will appear reddened) in order to reduce skin resistance. You must be fairly assertive when abrading the skin. Conform to the motto "abrade it like you mean it!" However, be careful not to cause the participant any pain while abrading. Ask them if they are experiencing any pain or discomfort while you are abrading the skin. Most often they will describe the procedure as "annoying but not painful." It works best if you abrade the forehead first and then attach the corrugator electrodes before abrading the cheek. This is because if it takes a while to attach the forehead electrodes, the abrasion may lose it's effectiveness on the cheek and you would most likely have to re-abrade the cheek again. The goal is to abrade each area only once. The paste should then be left on the skin for 1 ½ to 2 minutes, wiped off with a wet paper towel and then dried completely. Be sure you remove all the NUPREP skin prep gel before you attach the electrodes. Any residual gel on the skin will interfere with your ability to get good impedance readings.

For skin conductance, do NOT abrade the fingers with NUPREP skin prep gel or clean with alcohol because this will reduce the conductive properties of the skin. Just have them soap their hands with the cleansing cloth and dry their hands.

**Electrode Placement**

Respiration

The black Velcro strap is a strain gauge, measuring the degree of strain on the thorax (i.e., the girth of the thoracic circumference as one inhales and exhales). The gauge should simply be wrapped around the participant's rib cage, below the chest, near the bottom of the rib cage over the ventilatory muscles (see Figure 1). The gauge is plugged into the Respiration box in the system (hole # 7).

EKG and Heart Rate

We are using the standard limb lead II placement. Electrode ends are plugged into the 3M Red Dot #2239 Monitoring Electrodes. This is a disposable electrode with adhesive and solid gel. Place the electrode attached to the yellow cable just above the right wrist (on the inside of the arm) and the electrode attached to the orange cable just above the left ankle (on the inside of the leg). If a participant is wearing pantyhose, use the standard limb lead I placement: electrodes are attached just above the right and left wrists. The heart rate electrodes are plugged into the Isolation Bio2 Amplifier box of the system (holes # 8, 9, and 10).

Skin Conductance

We are using the bipolar placement for electrodes, involving the medial phalanges of two adjacent fingers—the third (middle) and fourth (pointer) fingers. (See Figure 2, bipolar placement). Make sure to use the participant's non-dominant hand. First, attach one side of the GRASS EWS-500 (25 mm or 1 inch) electrode washer to the electrode. Fill the electrode with EC22 paste for skin resistance and conductance, making sure it is smooth with no air holes. Remove the remaining adhesive backing from the electrode and place directly on the participant's finger. Repeat for the second finger. Wrap surgical tape around the two fingers to hold the electrodes in place. Place the participant's hand palm-down and rest it comfortably on the chair's armrest. The skin conductance electrodes are plugged into the Isolated Pre-amplifier power box of the system (holes # 11 and 12).

Temperature

Place the temperature probe on the participant's second (ring) finger of the non-dominant hand and put a cotton ball behind the temperature probe. Wrap surgical tape around the finger to secure the cotton and probe to the finger (hole # 6).

Facial EMG

We are measuring two separate facial muscle groups in the EMG psychophysiological assessment. One of them is the corrugator supercilii, usually involved in brow-pursing and frowning. The other, called the zygomaticus major, is mainly involved in smiling. Muscle movement will be assessed on the left side of each participant's face only.

Before hooking the participant up to EMG, TURN THE IMPEDANCE CHECK ON!!! The amplified output is passed to a circuit which lights a tri-color LED for each channel on the front of the amplifier unit. The display lights will be green if the impedance value is less than 5 kΩ, yellow for less than 10 kΩ, or orange for greater than 10 kΩ. After completely hooking up the participant, check the impedance level by looking at the color of the LED display (channels 2, 3, & 4). The EEG8 machine should be set on 100%. If the green or yellow lights are illuminated, you may proceed with the experiment. However, if the orange impedance light is illuminated, the electrodes must be removed, and the skin re-cleansed and re-abraded prior to reattaching the electrodes for EMG recording.

Because the electrodes will record the difference in electrical potential between differing areas of the same muscle, the facial electrodes will be arranged in pairs. Closely spaced electrodes (1 or 2 cm between them) is superior for observing the activity of single motor units, which is what we are concerned with in this study. Electrode placement must be done with great care. After the electrode sights are located and measured with the placement tool, any loose hair under the site should be moved. First, attach one side of the GRASS EWS-500 (19 mm or 3/4 inch) electrode washer to the electrode. Next, fill the electrode with EC60 electrolyte gel, making sure it is smooth with no air holes. To ensure the gel has no bubbles, you should "pack" the electrode a couple of times with the

gel. Scrap off the remaining gel so it will not interfere with adhesion of the collar. Finally, remove the remaining adhesive backing from the electrode and place directly on the participant's face in the pre-measured location. Repeat for remaining electrodes. Electrodes collars should be adhered to the electrodes so that the wires are positioned to the participant's left. Double check that impedance criteria are met (i.e., yellow or green lights) before securing with surgical tape to hold the electrodes in place.

NOTE: If you have a green light on the equipment after attaching the first electrode of a pair, this does not automatically mean the impedance for this electrode is good. The color of the LED display is meaningless until the second electrode of the "pair" is attached because this represents a "closed circuit." For example, if you have a green light after attaching the first electrode and then the LED turns orange after attaching the second electrode of the pair, do not automatically assume that the second electrode is the problem. It could just as easily be that the first electrode is not registering good impedance levels. Both electrodes should be examined and, if needed, removed, re-abraded, and replaced. In addition, be sure to replace the cap on the EC60 electrolyte gel tightly to avoid degradation of the skin conductance gel.

Body/Ground Electrode. There is a green electrode wire in the Body plug (hole #19) in the EEG8 box. Plug the end into a 3M Red Dot #2248 Monitoring Electrode with tape and solid gel. Place the electrode in the center of the forehead.

EMG Pair 1 in EEG8 Port 2 (A and B). Pair 1 will measure the corrugator supercilii muscle. The electrodes will be situated in a small arc above the eyebrow, beginning at a 60° angle to the facial midline (an imaginary line bisecting the face into two equal halves from the middle of the forehead down through the nose to the middle of the chin). The top electrode of the first pair (hole # 17) will be placed approximately 1 cm above the eyebrow and 2 cm to the right of the nasal midline (a perpendicular line bisecting the nose) when facing the subject (see Figure 3, letter A). Use the pre-cut placement tool to measure for standardized electrode positioning. The bottom electrode of the first pair (hole #18) is placed just lateral (next to) the first, following the arc of the eyebrow, keeping the electrode distance from the eyebrow about 1 cm. (see Figure 3, letter B, and Figure 4).

EMG Pair 2 in EEG8 Port 3 (F and G). Placement of the additional two electrode pairs will measure zygomaticus major activity. The top electrode of the second pair (hole #15) is placed approximately 2.5 cm from the base of an imaginary line drawn from the corner of the resting lip to the middle portion of the front of the ear (i.e., the condylion; see Figure 3, letter F, and Figure 4). Please use the pre-cut placement tool to measure from the corner of the resting lip for electrode placement standardization. The bottom electrode of the second pair (hole #16) is placed just posterior (behind) and lateral (next to) to the first (see Figure 3, letter G).

EMG Pair 3 in EEG8 Port 4 (H and K). The top electrode of the third pair (hole #13) is placed just posterior and lateral to the second electrode (see Figure 3, letter H)

and the bottom electrode of the third pair (hole #14) is placed just posterior and lateral to the other electrode in this pair (see Figure 3, letter K).

AFTER CONNECTING ALL EMG ELECTRODES, TURN THE IMPEDENCE CHECK OFF!!!!! The equipment is very sensitive and if the impedance check is not turned off at this point, the equipment WILL NOT record EMG properly and you would need to re-record the entire procedure.

Turn off the overhead light and turn on the lamp in the room prior to running the stimuli.

**Recording Procedure**

Once the equipment is attached securely, participants should be asked:

> *Are you comfortable? Is there anything causing you any discomfort? Please take a few breaths and try to relax for about 5 minutes before we begin the part of the experiment where you will see pictures. I'm going to be sitting in the next room at the computer making sure the experiment runs as it should.*

The computer hooked up to the psychophysiological system will be referred to as the "system PC" (the PC in the experiential chamber is where the participant sits); the second computer will be referred to as the "stimulus PC."

Turn Off Screen Savers

Turn the screen savers off from both computers.
Important or the screen saver will come in during the experiment!
On both the system and stimulus PC, double click on My Computer.
Select Control Panel and then Display.
From the Screen Saver tab, make sure NONE is highlighted.

Log Out of Novell on Both Computers

You must log out of Novell on both computers prior to running the experiment! This is because if the help desk should send an automatic message that the system is going down at a particular time, the experiment will be interrupted and you will have to start the experiment over from the beginning. This is not only time-consuming, but it is likely to make the participants unhappy if they have to repeat the psychophysiological procedures.

Turn on PsyStim2

From the desktop on the Stimulus PC, double-click on Shortcut to PsyStim2.exe.
Make sure you don't have anything else open that will show up on the bottom toolbar (e.g., Microsoft Word; Groupwise-Mailbox).
Go to the File Menu and select Hide Menu.

Turn on the CPU (Contact Precision Unit)

On the system, turn the big green switch to the on position (looks like |).

Start Psylab

Psylab is the software that runs our experiment and collects and analyzes our data.
On the System PC, right click the start button to bring up Windows Explorer.
Go to C:\CPI.
Double click on the icon for psylab7. Be careful to select this and not other icons with similar names.
PSYLAB Windows toolbar will appear.
Close the window for Exploring – CPI.
Left-click on the File Data menu and select Measure new data.
The File name is rohan12.pcc. Make sure exact file this appears in the box.
The next box says, "Enter SAVE EXPERIMENT DATA name."
Under "File Name:," type the name the data will be stored under. The suffix .0 must be included in naming all data files. In place of the *, we will call data by a 4-digit code. The first digit = assessment number (1 for pre-treatment or 2 for post-treatment). The last 4 digits = subject number, using all 3 spaces. For example, if subject number 39 is here for the pretreatment visit, data would be called 1039.0 If subject number 45 is doing the post-treatment visit, data would be called 2045.0
Click on OK.
A screen saying, "psym5 This application cannot be run in a window…" will appear.
Hit enter to continue.
Enter the subject number using 3 digits (e.g., 024).
Enter group number (1 = SAD from treatment study; 2 = Depressed but not SAD; 3 = normal control for SAD treatment study; 4 = SAD returning for 1-year follow-up; 5 = moderate seasonality from AU study; 6 = mild seasonality from AU study; 7 = nondepressed control from AU study).
For "Please enter number for SEQUENCE 1 TO 6," see the Psychophys Task Randomization Sheet. This refers to the order the blocks of photographs will be shown in. If it's the first assessment, put your participant's number in the next available slot and use the listed sequence number. If it's the second assessment, find your participant's number from the pre-treatment visit and use the listed sequence number for the post-treatment visit.
Hit enter.
The PSYLAB measurement screen will appear. Adaptation begins immediately.

Procedures During the Experiment

The first 2-minute period is used for adaptation to the equipment. While the participant is in the adaptation phase, you must calibrate the EMG equipment. On the PSYLAB system, in the center of the lower unit (this area is marked EEG8) go to "calibrate" and move the toggle switch to the "in" position. The switch should then be returned to the

"out" position. Calibration of the equipment SHOULD BE DONE WHILE IN THE ADAPTATION PHASE FOR EVERY PARTICIPANT. Then hit the unblock switch to ensure that the data is centered on the screen.
The following 3-minutes is used to record the participant's baseline responding. After adaptation and baseline, the PSYLAB Measurement screen will pause and print, "PRESS F1 to continue." The experimenter should then say to the participant:

*Please fill out the top questionnaire measure (#0) to rate your mood. Tell me when you have finished.*

After they finish, say:

*We will now begin showing you pictures. Remember to relax and imagine what it would feel like if you were actually in the picture. Imagine what you would be feeling and thinking if you were really there.*

Press Function Key #1 (F1) to resume the experiment. After presentation of each block of slides, the "Press Function Key #1" will appear. Be sure to watch for this to keep the flow of the experiment going and to avoid a lot of down time for the participant. After each block, tell the participant to complete the next measure in the pile (this will be the pleasantness rating scale and the POMS; #1). At the end of the experiment, you should have 7 completed questionnaire measures, including 6 sets of pleasantness ratings and 7 POMS ratings. Each set should be marked with the subject number and date in the upper right corner.

At the end of the experiment, the PSYLAB Measurement screen will disappear and the Psylab toolbar will return on the screen.

NOTE: BEFORE UNHOOKING THE PARTICIPANT, YOU MUST PHYSICALLY VIEW THE AMPLIFIED DATA TO ENSURE YOU GOT GOOD READINGS!!!!

Click on File
Click on "View measurement data"
Enter the participants number (e.g., 1 = pre-treatment, 2 = post-treatment assessment followed by the participant number). For example: the pretreatment for participant number 080 would be entered as 1080 in order to view the data you just recorded.
When the data trace comes up, view the data to make sure it looks good. If you are unsure what a good trace looks like, check the file 1000 or 000 to see what a good trace looks like.
Double-check the "data" window to ensure you have the correct participant number, group number, and randomization sequence. The number of files recorded will vary from participant to participant. However, the "data files" will usually have around 500 or so files, whereas the "binary files" will usually have around 1500-2000 files. Just check briefly to ensure you have something similar to these numbers.

**Clean-Up**

After presentation of the sixth and final block of stimuli and completion of the POMS and pleasantness ratings tell the participant:

> *This completes the portion of the experiment where we measure your reactions to photographs. I'm now going to remove the disks and straps so you can continue with the other procedures.*

Then carefully remove each electrode so as not to cause the participant any discomfort. Wash the areas again gently with a facial cleansing cloth in order to remove any leftover EC60 electrolyte gel or EC22 paste. Make sure you remove everything—respiration strain gauge, 2 EKG electrodes from ankle and wrist, 2 skin conductance electrodes on fingers, 6 facial EMG electrodes, and body/grounding electrode on face. Following removal of the electrodes and cleansing, say to the participant:

> *I thank you very much for your participation in this study. I know that sometimes these situations can be uncomfortable, but I really appreciate your time and effort. Is there anything else I can answer for you before you leave today?*

The electrodes should then be thoroughly cleaned out with a Q-tip.

To bring the menu back on the Stimulus PC, hit the control (Ctrl) key with PsyStim2 open.

On the system PC, close the Psylab window.

**Back-Up All Data**

NOTE: YOU MUST BACK-UP ALL DATA AFTER RECORDING BY SAVING IT TO THE "Q" DRIVE!!!! This will be our only back up data in case something goes wrong with the system computer so make sure you do this after each participant you run.

Login to Netware (right click on red "N" at lower right hand corner of computer)
Right click on "start" menu
Open "explore"
Click (+) next to "Q" drive to display folders
Scroll up to the top and click on the "C:\CPI" folder
Bring "KTL Psychophys Data" folder on "Q" drive into view in the left window
Go back to the C:\CPI folder and highlight all files with participants number (highlight the first file, hold down the "shift" key and then hit the right arrow key until all files with the participant's number are highlighted)
Drag and drop the files in the "KTL Psychophys Data" folder (NOTE: There will be several hundred files so make sure you copy them all)
Close out of the program

*Figure 1*. Placement of respiration strain gauge.

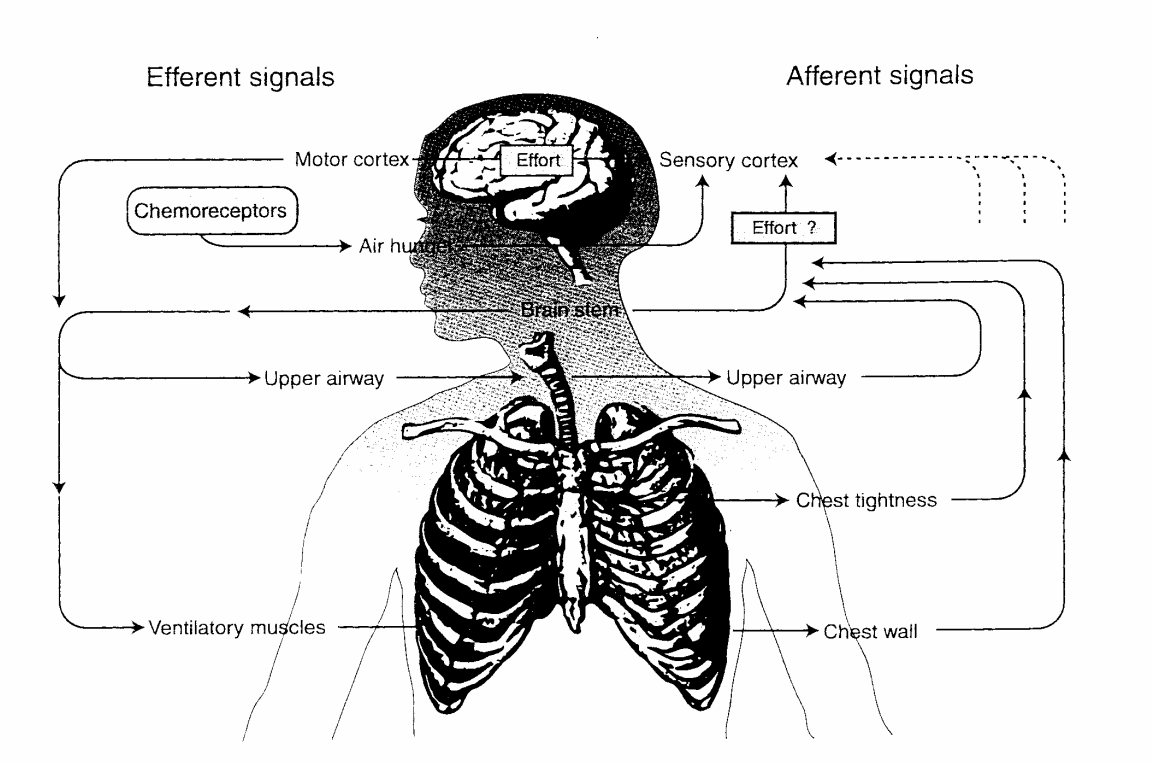

*Figure 2*. Placement of skin conductance electrodes.

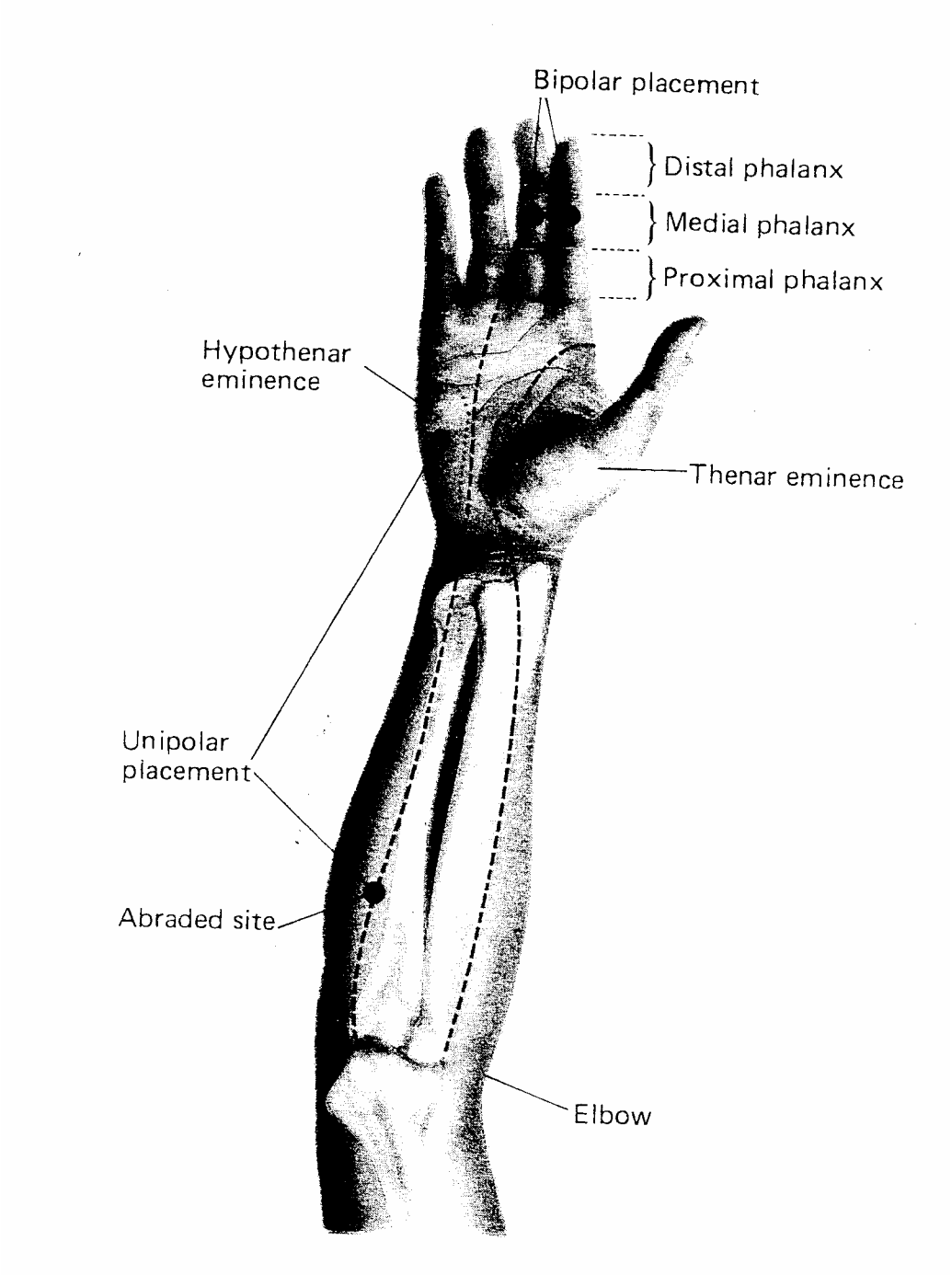

Figure 3. Placement of surface facial EMG electrodes.

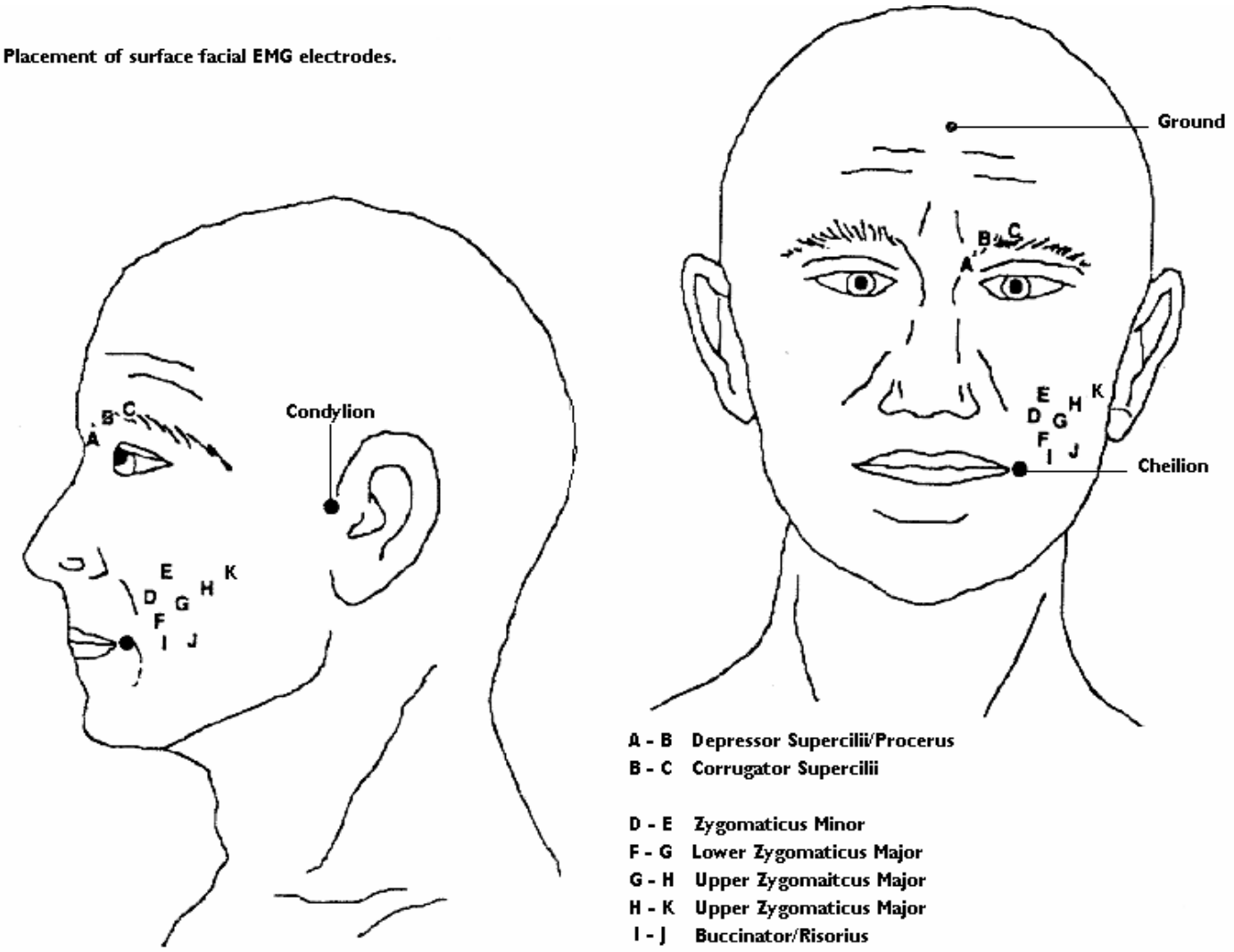

*Figure 4*. Anatomic illustration of facial musculature.